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THE VETERINARY JOURNAL

A Monthly Review of Veterinary Science.

Editors for Great Britain and Ireland:—

FREDERICK HOBDAY, F.R.C.V.S., F.R.S.E.,
FORMERLY PROFESSOR IN THE ROYAL VETERINARY COLLEGE, LONDON.

GEO. H. WOOLDRIDGE, F.R.C.V.S.,
MEMBER OF THE ROYAL IRISH ACADEMY,
PROFESSOR OF HYGIENE, MATERIA MEDICA AND THERAPEUTICS IN THE ROYAL VETERINARY COLLEGE, LONDON.

Editor for Australasia:—

J. A. GILRUTH, D.V.Sc., M.R.C.V.S., F.R.S.E.,
DEAN OF THE VETERINARY FACULTY, UNIVERSITY OF MELBOURNE, AUSTRALIA.

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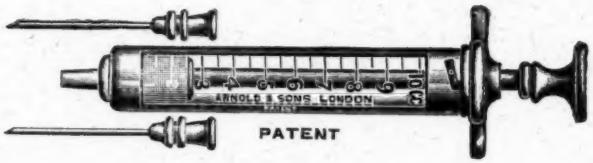

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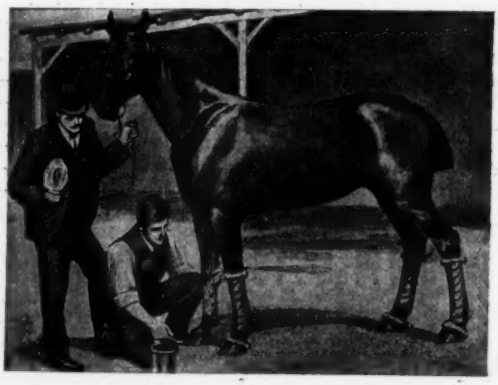

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THE

VETERINARY JOURNAL

APRIL, 1910.

THE LATE PRESIDENT OF THE ROYAL COLLEGE OF VETERINARY SURGEONS,

SAMUEL LOCKE, M.R.C.V.S., 1845-1910.

ALL our readers will regret to hear of the death of the esteemed President of the Royal College of Veterinary Surgeons, which took place on March 9, at Manchester, in his sixty-fifth year. Apart from the personal loss which many of us feel, Mr. Locke will be sorely missed at the Council Meetings, where he had proved his marked ability in smoothly directing the work of our representatives under difficult circumstances. No man has ever had the welfare of his profession more at heart than Mr. Locke, who practically died "in harness," as he would have wished.

Born at Salford, Mr. Locke served a pupilage with the late Mr. John Lawson, and, proceeding to the Glasgow Veterinary College, he took his diploma in 1869. For many years he was a member of the Lancashire Veterinary Medical Association, of which Society he officiated at different times as President and Secretary, while for nearly twenty years, right up to the time of his death, he acted as Secretary of the National Veterinary Benevolent and Mutual Defence Society. He became a member of the Council of the Royal College of Veterinary Surgeons in

1895, and was a Vice-President in 1900, 1904, 1906, and 1908. Last year he attained the highest honour his profession could bestow upon him and was elected President. During his term of office his exercise of great tact and business capabilities more than justified the confidence reposed in him, and it is a great loss to the profession at large that he has not been spared to complete his term of office.

In addition to the duties involved by a busy practice and his various labours on behalf of his profession, Mr. Locke was able to find time for much public work. He was a prominent member of the Manchester City Council, and was for many years an overseer for South Manchester. It was largely due to his efforts that the excellent system of meat inspection conducted by properly qualified veterinary officials was introduced. He was a trustee of the Manchester Royal Infirmary, and Vice-Chairman of the Manchester Adult Deaf and Dumb Institute. Mr. Locke was also a prominent Freemason, being particularly interested in the Masonic charities. He was a Past Master of the Chorlton Lodge, and a Past Provincial Grand Deacon of West Lancashire.

This is the first time that a President of the Royal College of Veterinary Surgeons has died during his year of office.

Editorials.

VETERINARY EXAMINATIONS AT DOG-SHOWS.

In the course of his very excellent contribution on Distemper in this issue of the Veterinary Journal, Mr. Livesey incidentally draws attention to the question of veterinary examination of dogs at shows. Mr. Livesey says, "The present veterinary examination at dog-shows is an absolute farce." While we are not able to go to this extreme, we must admit that the examination referred to very frequently indeed leaves much to be desired. The conditions under which a veterinary surgeon has to examine dogs before they are admitted to shows render it absolutely impossible for the examination to be as perfect as it should be. It is absurd to expect any man, no matter how expert he may be, to examine the number of dogs he usually is expected to examine in the time allotted for the purpose. It is usually only dogs in the early stages of the disease, or in the convalescent and still infective stage, that are likely to be overlooked; and these are cases in which a diagnosis is often extremely difficult, especially when the owner is aware of the state of affairs, and does all in his power to hoodwink the examiner. It is a curious trait in the character of a number of dog-breeders who, while complaining bitterly of the risks of infection at dog-shows, do not act as squarely as they should do themselves. How frequently we find doggy people wiping the eyes of their dogs before the veterinary surgeon can see them in order to avoid the possibility of a catarrh being diagnosed as distemper. Veterinary surgeons know quite well that a conjunctival discharge may be due to many other causes than distemper. On the other hand, if the case should be one of distemper, surely it would be better for all concerned that the dog should be sent home—better for himself and his owner as well as for the other dogs present. We believe that many of the dogs that have died of distemper after shows were already infected before they were taken to the show, and if the veterinary examiner had only been assisted instead of misled by the owner, they would have been taken home and properly nursed at the most critical time and would have

recovered ; and, moreover, the lives of many other valuable dogs with which they were brought into contact would have been saved also.

We admit, then, that numerous dangerously infective dogs are allowed to be exhibited at shows, and the question arises as to how it is to be prevented or minimized. We believe there is a solution to the problem, but it can only be attained by the co-operation of all concerned, viz., the organizers, the exhibitors and the veterinary surgeons. The fact that there is a veterinary examination at all prevents obvious cases of disease being brought to the show, but the less obvious ones are brought in the hope that they may be overlooked in the hurry of examination.

In the first place, then, there should be a larger number of veterinary inspectors appointed in order that a proper examination may be possible. No veterinary surgeon should examine for admission any dog belonging to one of his own clients. Exhibitors should be informed of this fact, and they should assist in examination instead of attempting to cover possible symptoms of disease. An additional veterinary surgeon should be appointed as a referee to decide on cases of dispute, and his decision should be final. In the case of smaller local shows when only one veterinary surgeon is required, he should not be the local veterinary surgeon. This would prevent local jealousies, and also guarantee an impartial decision as to the fitness of exhibits.

As to the question of payment of veterinary inspectors at shows, we are of opinion that a fee should be paid as a matter of principle. If any veterinary surgeon preferred not to be paid for his duties in this connection he could return his fee to the show funds in the form of a donation. On the other hand, the veterinary surgeon is acting as a specialist for the protection of the owners of more or less valuable animals, and his services should be adequately recognized.

DEGREE OF DOCTOR OF VETERINARY SCIENCE.

In view of the recommendation passed by the last International Veterinary Congress, it is interesting to note that the statutes of the Melbourne University provide both for the degree of Bachelor and that of Doctor in Veterinary Science. When the question as to whether the term "Veterinary Medicine" or the term "Veterinary Science" should be employed was discussed by the Faculty, it was unanimously decided that the latter being more comprehensive than the former should be recommended to the Senate, and this was approved.

The regulations state that candidates for the Degree of Bachelor "shall subsequently to their matriculation pass five examinations and complete five years"; that "Candidates for the degree of Doctors must be Bachelors of at least two year's standing in the University of Melbourne"; and that "Candidates may proceed to the degree of Doctor by examination or by presentation of a thesis."

It is also provided that Members of the Royal College of Veterinary Surgeons who have been resident in Australia or New Zealand for a period of twelve months prior to the year 1912 may, on the recommendation of the Professrial Board, be admitted to the degree of Bachelor on writing a thesis to the satisfaction of the special examiners appointed; two years later, of course, they are qualified to proceed to the degree of Doctor, either by examination or by thesis.

A further concession has been made to heads of State Veterinary departments in Australia and New Zealand, and to Veterinary Surgeons engaged in teaching at the University, by a temporary Regulation permitting them on the recommendation of the Faculty to proceed to the degrees of Bachelor and of Doctor of Veterinary Science simultaneously, by presentation of thesis. Already Messrs. S. S. Cameron, Chief Veterinary Officer of Victoria, Professor Gilruth, and Mr. W. T. Kendall, of the University, have complied with the conditions and had the degrees conferred, while Mr. Sydney Dodd, Chief Veterinary Officer of Queensland, has been notified that his thesis has been accepted, and that the degrees will be conferred in due course.

General Articles.

DISTEMPER IN DOGS.

By G. H. LIVESEY, M.R.C.V.S.
Hove.

In contributing a paper on distemper I feel sure that some notes on one's clinical experience will be probably more acceptable than a lengthy review of what has already been written, or a criticism of various writers' statements as to the cause of this terrible scourge.

Although it is so widespread and so well known, distemper is one of the diseases which is least often diagnosed in its early stages. One of the greatest difficulties in the way of successful treatment is the inability of a large number of veterinary surgeons and of the public to diagnose distemper until the disease is well established or complications have supervened. So long as distemper is unrecognised until marked nasal and conjunctival catarrh are present, so long also will the death-rate of the disease remain at its present high figure.

The general public do not know the early signs of distemper, and we may feel fairly certain that a very great number of veterinary surgeons now in practice are not much wiser. For, as proof of this, one has only to attend almost any large dog show and carefully look over the young dogs and puppies. As dogs suffering from distemper are not supposed to be received at any show we are forced to the conclusion that these cases have been unrecogized. Moreover, after every large show, and especially in damp weather, we always hear of a number of cases of distemper "which has been caught at such and such a show." At one large show in London I saw several cases. I have been told by other veterinary surgeons that many so-called doubtful cases have to be admitted by them to shows as a matter of diplomacy, for the public and breeders who pay good entry fees must not be offended, and a man must not make himself so unpopular as to risk his future appointment. It is a most difficult matter to decide quickly that a young dog has not got distemper, and it is an impossibility for one man to examine say two hundred dogs in a couple of hours and do himself and his profession justice. For this and other reasons I have now refused to act as veterinary surgeon to any show. I know some members of our profession who depend for their diagnosis upon the presence of "a fleck of matter in the corner of the eye," and a rise of temperature and depression. I feel sure that such gentlemen, if they act for shows, must pass in a lot of incipient cases. I have often seen owners wiping out the corners of their dogs' eyes

before showing them to the examining veterinary surgeon, and how many of these latter gentlemen have the time, even if they have the inclination, to take temperatures? Moreover, temperatures recorded amid the turmoil and excitement at the entrance of a show are notoriously unreliable. Apart from shows, however, it is still a difficult matter to say with certainty that a young dog is quite free from distemper infection. A great deal must be taken into consideration in giving an opinion. Of course, if he has been exposed to infection, only careful observation for ten days or a fortnight will decide whether the disease will develop or not. When large numbers of dogs are kept together it is almost impossible for any to be quite free from infection; the older the dog, however, the less likely is he to show any clinical symptoms—still he may be capable of carrying infection to others, and especially to young whelps. I cannot call to mind any place that I have seen where a large number of dogs are kept together in which I have not found clinical evidence of distemper. Very few of the hunt kennels are ever free from infection—dogs' homes never. One cannot help being struck with the fact that though many young foxhound whelps die of distemper when out at walk, frequently the greatest mortality occurs when they come in from walk and are for the first time introduced to kennel life. A number of young dogs are freshly brought from a life of comparatively perfect freedom, crowded together on ground and in kennels which are not free from the taint of previous year's infection—is it to be wondered at that so many suddenly develop the disease and die?

Many veterinary surgeons in practice declare they will not take distemper cases into their infirmary. Yet they do so nearly every day. They may not take in cases where there is a profuse discharge from the eyes and nose, but they do not seem to fear the introduction of that husky cough or that intractable diarrhœa. If one excludes distemper cases, the number taken in is diminished by more than half. I should advise any veterinary surgeon who was intending to make a speciality of dogs to either keep quite a small hospital and rigidly exclude every case in which there is room for any doubt, or keep a large infirmary and admit all and sundry. If he keeps a large establishment the difficulty of rigidly excluding distemper is almost insurmountable.

Distemper manifests itself in so many and various forms, the symptoms are so numerous and in our present state of insufficient knowledge each case seems to call for its own special treatment. In a bad outbreak in a district where several large kennels are situated,

it will show itself in all ways and one has apparently to treat all the more common ailments that the dog is heir to, while in reality only encountering the one disease. From the above remarks one would naturally suppose that distemper is a perfect godsend to the veterinary surgeon. It may be so to those who prescribe for each series of symptoms as they arise and lead their clients to believe that the dog is suffering from either cough, eczema, diarrhœa, jaundice, gastritis, epilepsy, pneumonia, bronchitis or common cold, but to those who in all these ailments see the one prevailing infection, with any secondary infections merely supervening as complications or sequels, the treatment will probably be of the simplest. For I believe those cases are most successfully treated where least medicine is given. I do not mean that the valuable assistance of medicine is not to be made use of, but that if one wishes to save one's patients or at least obtain a good percentage of recoveries, medicine should not be given unless for some very definite purpose, and when it is not confidently expected to assist in relieving the symptoms. The practice of giving "something in a bottle" is to be strongly condemned. If our patient does not require it, we have no right to prescribe it, as it may endanger his chance of recovery, by nauseating him and making him resent the administration of nourishment, which, according to my own experience, is of far greater importance than any drug in the pharmacopœia. This is especially true in regard to cats. If you have learned the secret of getting sick cats to take an adequate amount of nourishment you have mastered more than half the difficulties of treating them when ill. Of course if one only sees a very few dogs, it may seem desirable to make the case "worth attending" by the prescription of sundry bottles of medicine. A man should honestly try his utmost to cure the cases put under his care, and if he can do so best by refraining from prescribing medicine, no monetary consideration should tempt him to do other than what he considers best for his patient. Writing from my own experience, I say that in our present state of knowledge undoubtedly the secret of success in treating distemper is first to see the patient early and be able to diagnose the presence of the disease before complications have set in; the treatment is then very simple. Second, to take no risks, but be exacting in having the greatest care taken to protect the patient from sudden changes of temperature, damp, and from infection from other dogs; in fact to isolate him. Thirdly, have him nursed as if he were a human being. The greatest care is needed when the dog is apparently quite well, as a relapse is always far worse than the original disease.

How are we to recognize distemper when a case comes under our notice? This is a difficult question to answer, and one on which I can only venture an opinion.

Symptoms.

From a clinical point of view the disease in its simplest form is undoubtedly the most difficult to recognize; when complications ensue, the disease is manifest to the layman, and we, as a profession, should be able to make our diagnosis before the layman realizes from what disease the dog is suffering.

In the simplest form, the symptoms of distemper are essentially those of septicæmia. After infection there is probably a slight rise of temperature and there may be a little languor, but this is so slight that it is seldom, if ever, noticed. About five to fifteen days, which I believe to be the incubation period in the majority of cases, there is probably a little diarrhœa or vomiting. This may be very slight also, and calls for no comment on the part of the owner. The dog may refuse his meals for a day, but this is a symptom common to many minor ailments, and causes no alarm. If, however, all the skin, and especially the skin of the abdomen, flanks, elbows, thighs, the lips and ears be carefully searched every day there will nearly always be found one or two pustules or the scab or scar left by a pustule which has already become dried up. These pustules are quite distinctive, and are peculiar to distemper. They form rapidly in about four hours, having the appearance of a small rounded pink or red lump, something like a small heat-bump in man. In another two hours or so the swelling becomes translucent at its summit, and contains a thin clear fluid. The fluid rapidly becomes of a creamy consistence, and the swelling becomes a conical pustule with an irregular outline, sometimes circular and sometimes oval, and varying in size from that of a pin's head to that of a horse bean. They become ruptured in from six to twelve hours after maturity, the contents drying into a brownish scaly scab. If this is picked off, there is left a pitlike depression, and if the pustule has been large, the healing of this depression leaves a distinct scar.

These vesicles may be discrete or confluent, there may be only one, or there may be hundreds. In the earliest stage it is commonest to find only a few. This is the primary rash of distemper, and unless carefully searched for will probably be missed, as it is evanescent, the pustules often forming and drying in twenty hours. If the vesicles have not been noticed except when pointing with purulent material, they may often be mistaken for insect bites, especially those of the

harvest bug, or the scab may be mistaken for scurf, and especially so if the dog has recently had sulphur ointment applied for some skin disease, or if he has lice on his skin.

Particular attention is drawn to this eruption, as in the early stage, at least, it is the only certain evidence which we have that the dog has distemper infection in his system. No dog showing such an eruption ought to be allowed to enter a show on any consideration. When once it has been noted, the patient should be kept under observation. No further symptoms may be noticed, but the dog can hardly be considered to be safe or free from further distemper symptoms for six weeks. This may seem an abnormally long period of quarantine, but I am satisfied that the infection may remain latent in the system for a long period, ready to show itself whenever the vitality of the patient is lowered and his power of resistance weakened.

If now the disease progresses a regular sequence of symptoms is noticed. The most noticeable is want of condition. The patient does not thrive, and, in spite of good feeding, not only fails to put on flesh but even loses weight. His temperature is sometimes normal, and sometimes subnormal, his heart is slow or intermittent, he loses his happy look, and is more or less dejected, though at times he may be roused and play like a healthy dog, but he soon tires and spends more hours than usual sleeping.

His appetite is capricious and occasionally he vomits after food and may have a husky cough. His bowels do not act with regularity. He may be constipated for one or two days, but generally has an intermittent rather intractable diarrhœa. The coat loses its gloss and becomes harsh and staring, while the skin is more or less leathery and often carries an unpleasant doggy odour and often is slightly moist. This condition may continue for several weeks. To most people the dog is merely a "bad doer" and receives no particular attention.

The eyes nearly always show a minute speck of greyish-coloured mucoid discharge at the inner canthus, especially in the mornings, but this discharge is not purulent like the discharge seen in advanced cases. The nose is sometimes moist, and there is seldom any noticeable discharge from it. The lining membrane of the ear is nearly always more moist or greasy than usual owing to a slight seborrhœa, and the dog is now particularly liable to parasitic canker. He easily becomes the host of various parasites, both internal and external, his lowered vitality rendering him less energetic in avoiding or killing skin parasites, while his depraved ppetite leads him to eat filth and with it infect his stomach or bowels with worms.

If the dog be now exposed to cold, damp or fresh infection, distemper develops quickly. The temperature is raised a degree or so, but may rapidly fall again, appetite is lost and possibly there is rigor or even a slight convulsion. Emaciation progresses very rapidly, and the dog becomes semicomatose. There is a purulent discharge from the eye, but only very small in quantity, the nose is dry and hard and often covered with a gummy scale. The mouth is dry and the lips, tongue and pharynx often ulcerated, while the odour of the breath is very offensive. Diarrhœa is frequently present and the fæces are often streaked with blood. The heart sounds are generally partially obscured by pericardial effusion and small areas of broncho-pneumonia may with difficulty be located by careful auscultation. At this stage—about the fifth to ninth since the second invasion—the patient dies of exhaustion and death is frequently ushered in by convulsions or persistent coma.

At any time during this secondary stage of the disease a fresh (secondary) eruption of vesicles may be seen. The pustules are less frequently discrete and they are generally more numerous and do not tend to heal so quickly. When ruptured the sore may ulcerate and the ulcer may burrow deep into subcutaneous structures.

If recovery takes place, either on account of good nursing or from the *vis medicatrix naturæ* there is a gradual amelioration of the patient's condition shown first in the general appearance. The eyes are brighter, and the dog takes interest in his general surroundings, ulcers heal, appetite and bodily power returns, and last of all the discharge from the eyes and the diarrhœa cease.

The symptoms I have recorded are those commonly seen in a case of simple uncomplicated distemper. In some cases these symptoms, though present, may be partly obscured or lost sight of owing to less common manifestations of the disease being more strongly in evidence. For instance, before the owner realizes the presence of distemper, he may find his dog suffering from icterus. This often proves very troublesome, and in packs of hounds causes great mortality. The absorption of the bile does not arise from blocking of the bile duct, or from overloading of the gall-bladder, for at many *post-mortems* I have invariably found the duct quite patent, and the gall-bladder seldom more distended than in a healthy dog, moreover, the duodenum has shown little or no sign of catarrh. Another condition, not always seen, is deep coma from the commencement of the second infection. This is often accompanied by a very subnormal or very high temperature.

In the former case the dog generally dies without a struggle and without regaining consciousness, while in the latter the coma often gives place to violent convulsions, which nearly always end fatally. Another condition less frequently seen is a state of hypersensitiveness. This is a manifestation of infection of the spinal cord, and may indicate myelitis or spinal meningitis. Sometimes it causes regular convulsions, but more often merely hyperæsthesia and tremor of various groups of muscles, especially those of the legs and head. It is commonly followed by chorea or paralysis.

If the case is protracted, certain other symptoms are fairly common. The eyes, and especially those of the short-nosed dogs, are liable to give great trouble. The conjunctivæ become suddenly highly inflamed, tears flow abundantly, and there is marked photophobia. The inflammation has a great tendency to spread to the deeper structures, and there may be a panophthalmia followed by loss of the eye, or the cornea may ulcerate, commonly at the centre or upper internal portion. The ulcer may be followed by staphyloma, it may penetrate an anterior chamber, and the aqueous humour may escape and even carry with it part of the iris. The ulcer becomes connected with the sclerotic by arborescent vessels, the cornea becomes opaque, and the sight is impaired or lost. Another symptom, not always met with, is the development of subcutaneous abscesses. These are very troublesome, and if not quickly evacuated may cause death from septic absorption. Even when well drained and dressed they often take long to heal and leave ugly scars. Frequently, when occurring in numbers, they may at first sight be confused with follicular mange, and they are often followed by complete temporary loss of hair over nearly all the body. Probably there is no disease which, running concurrently with distemper, does not become increased in severity itself, while at the same time emphasizing each symptom of distemper. When a dog with distemper becomes infected with another disease or with distemper from another source, the original infection becomes exalted. This is shown by markedly rapid emaciation and a temporary rise or fall of the body temperature, an increase in the severity of the symptoms already reviewed, together with a manifestation of the symptoms of the complicating ailment. The commonest complication is common coryza, or nasal catarrh, giving the name "Snuffles" or "Snifters" to the disease so recognized. The other common and very fatal complication is lobar or purulent pneumonia, easily distinguishable from the slight broncho-pneumonia so frequently seen in the secondary stage of simple distemper.

Diagnosis.

In the diagnosis of distemper we meet with our greatest difficulty in the early stages. When the disease is well established diagnosis is easy, but proportionately as symptoms increase making diagnosis easy, so treatment becomes more complicated. For simple and successful treatment in a large number of cases it is essential to make an early diagnosis and to keep the patient under observation.

In all cases of dogs under 2 years old which are brought with a history of malaise, diarrhœa, loss of appetite, suspect distemper and carefully examine the eyes for a watery or mucoid discharge and the skin for any pustule or trace of a pustule.

In all cases of puppies under 9 months brought with a history of vomiting, convulsions, or diarrhœa, especially if there is loss of appetite, suspect distemper and search for the eruption. In this case the majority of people would probably say suspect worms. No doubt worms do cause a lot of trouble in puppies, but thousands of wretched little puppies have their lives made miserable and their health shattered by continual "worming" when the real cause of their not thriving and of having so-called "puppy eczema" is not worms, but distemper. The emaciation caused by distempter is general, and the belly is usually pinched up. The skin nearly always has a peculiar sickly smell, is often moist, and the pustules, if present, are easily seen and generally discrete. Emaciation from worms is never so rapid, and the growth of the skeleton is not so much interfered with, the belly is distended unevenly, and the stomach and duodenum can often be felt hugely dilated under the skin and muscles on the left side. The pup is, in fact, pot-bellied. The skin is dry and often scurfy, or there may be a slight erythema, but worms do not cause pustules on the skin.

In any case of a dog brought with the history of a husky cough and loss of condition after having been to a show, or after being at a veterinary surgeon's kennel, especially the larger of these establishments, one should certainly suspect distemper. Not only is the disease so common and prevalent, but such little real care is taken to exclude infectious cases that wheresoever many dogs are gathered together there surely will distemper be found. There are many breeders and many who regularly show who say there are diseases named "influenza" and "show-bench cough," and they admit that they are contagious; this is distemper proper, and it is only the unwillingness of breeders to admit that they have the real disease in their kennels that makes them find for it some fancy name.

In any case where in a young dog there is progressive emaciation, unthriftiness, lack of appetite over a considerable period, together with an abnormal readiness to sleep through most of the daylight hours—suspect distemper. In these cases one may have to wait a long time before having one's suspicions confirmed, but when confirmatory symptoms arrive the case often runs a rapid course, and frequently ends fatally—often in coma or convulsions. This is the so-called suppressed distemper, and is one of the most difficult forms to diagnose and to treat.

In practice one has not to treat many cases other than distemper or arising out of distemper—surgical cases excluded. Even the fact that a casual patient is under 18 months old should put a veterinary surgeon on his guard lest he overlooks this prevalent disease. Often a puppy is brought with a history of having suddenly gone mad, or having a screaming fit in the street, and it is generally supposed that he has worms. Such cases, if kept under observation, only too frequently develop other symptoms, such as rapid emaciation and a discharge from the eyes. In these cases the eruption is not always seen, and the convulsions or emaciation and depression are the only symptoms of a bacterial intoxication of the system.

In examining a dog rapidly in order to diagnose the presence or absence of distemper—a duty required of many of us at shows—it is best to first look at the dog's eyes, close the lids, and press the eye inwards, opening first the lower lid while the membrana nictitans is still across the eye and searching for any discharge that may be hidden in the pocket of the inner canthus. Do not fail to remember that most doggy people at a show wipe their dogs' eyes carefully before showing them to the veterinary surgeon at the door. Next look at the skin of the belly and thighs and under the elbows and inside the ear, searching for any trace of a pustule; notice his general condition. If there be any doubt put him on one side and examine him more carefully when there is more time for the job. It is well to become well acquainted with the pustular eruption and the remains of it, and it is unlike other skin affections, and when once seen it is easily recognized.

Do not be led astray by anything that may be said by the owner; you will frequently be told that the dog has already had distemper, but that is no reason that he should not contract it again, especially from a fresh source. You may be told that the dog had distemper within the last three months and therefore he cannot now be suffering from it as he got it quite well. In such cases remember that relapses are very common, and that a dog recovered or recovering from the disease is

very subject not only to relapse owing to want of care on the part of the owner, but also to fresh infection from another source.

The value of the thermometer in diagnosing distemper has been unduly overrated by most writers. If you depend upon your thermometer for the diagnosis of distemper, I believe that a vast number of your cases will go undiagnosed. If a temperature chart be kept of a case under observation, the rise or fall of the body heat is often of great service in enabling one to give a prognosis, for, while at a single observation the height of the body temperature is no indication of the presence or absence of distemper, a continued series of such observations in a case where the disease is known to exist records the advent of complications or the beginning of new infections from a fresh source.

If these statements be accepted, it will be at once apparent that to reject a dog at a show merely because he has a slightly raised temperature is neither fair to the dog nor his owner. At all times one must not allow one's perception of the disease to be obscured by the many other symptoms which may arise during its course; one may look beyond these at the disease itself.

Prognosis.

The prognosis of distemper is always guarded. The most hopeful cases often end fatally, while on the contrary many apparently hopeless cases recover. The prognosis varies somewhat with the breed of the patient; it is never good in any short-nosed dog; in Japanese it is always grave. Foreign dogs are generally more seriously affected in this country than natives; in any dog which is inbred it is not so hopeful as in a dog which has been bred from divergent stock—such are more prone to affections of the nervous system. Pampered animals suffer more than those which have had an early struggle for existence, and those dogs which have had a previous severe attack acquire a certain immunity; but this is not an invariable rule, for even they may succumb to a fresh and virulent infection. A protracted case is never so hopeful as one which runs its course quickly, and a protracted case in which complications set in late generally ends fatally. A sudden rise of temperature after a long spell with the temperature normal or subnormal generally heralds death from pneumonia or convulsions, while a continual loss of heat shows profound depression of all the vital functions with coma and exhaustion. A sudden fall generally foreshadows death from syncope.

A profuse eruption may be taken as a good sign, for it must be

better to have such purulent material out of the system as soon as possible. For this reason a sudden cessation of all eruption, or a sudden ceasing of diarrhœa, are not good signs, and often precede convulsion and other symptoms of nervous intoxication. Any rapid change, in fact, in the symptoms is not to be welcomed; even a sudden appetite for solid food after a long period of forced feeding. One should be on one's guard against satisfying this craving, for such a meal is often followed by convulsions.

In this disease there should be no need to despair of any case so long as life and consciousness remain, but the longer recovery is delayed the less likely is it to be established.

Treatment.

In the first place, there is no specific treatment for the disease, and well it is so, for the public would soon learn its nature, and nearly half of our present dog patients would not come under our hands.

It is a pity the public does not realize the fact that all the so-called specifics and cures so freely advertised are practically useless. It is a pity also that many veterinary surgeons do not realize that any single drug or combination of drugs cannot be considered suitable for all cases. I have seen some who have one bottle of pills, or a mixture labelled "Distemper," and each and every case is dosed from that bottle, no matter what symptoms may be presented. In fact, the patient must be made to conform to the attendant's routine, treatment or fad.

For those who take dog practice seriously distemper is a source of great trouble, and the care of the patient a continual anxiety; for the most hopeful cases often die in spite of all that may be done for them. Having, during the last two or three years, seen an exceptional number of cases (for distemper has been very severe in my district), I have become almost a fatalist as to a dog's chance of recovery. Many dogs will recover without treatment, and others will die, no matter what is done for them, and others, I fear, get well *in spite of* the excessive treatment they receive. A great deal, of course, depends on the breed and constitution of the patient, but more, I believe, on the virulence and source of the infection. In one outbreak nearly all will die, in another deaths will be very few. So also deaths may occur frequently in one family, while all the members of another survive. It is hard to realize this when attending single cases, but it is very noticeable in large outbreaks and in epidemics.

I say nothing as to preventive treatment. I leave that to those

who profess to know about it. I do not; and I maintain that until we can be absolutely certain of the cause of the disease we cannot obtain any preventive agent that is worthy of consideration. I have tried a large number of so-called preventives, and have found them of very doubtful value, if not useless. Good health and careful avoidance of dog shows and large kennels is still the best safeguard against infection.

To my mind, the secret of such measure of success as we may hope for in treating distemper depends upon three main things; (1) The early recognition of the disease: (2) the isolation of the patient; (3) good nursing, and the use of appropriate medicine. This may not necessarily be true in single cases, but it is emphatically true when one reviews the course of several hundred cases.

With regard to my first point—the early recognition of the disease—I fear that I am at variance with a large number of the profession. I maintain that the presence of the cutaneous eruption is absolutely diagnostic of the disease, even in its earliest stage. This eruption is not considered diagnostic by many, while others, though admitting it to often be suggestive of distemper, do not consider the patient infectious, and do not think it necessary to take any precautions for the patient's welfare. One has only to examine the dogs at any show to see that what I say is true.

I fully realize that what I recognize as distemper in its early stage is not believed to be distemper at all by a very large number of veterinarians. I have had ample proof of this, and am continually getting more. Dogs suffering from distemper are brought to me from all parts, which have been treated for various complaints—complaints bearing any name but distemper, and I am satisfied that the veterinary attendant in each case has been treating symptoms without searching for their true cause, giving the name of some special disease to each symptom as it has arisen. In this way the disease is allowed to get established in the dog before proper precautions are taken; in this way the disease is often spread through large kennels. The commonest cases that I see which have been unrecognized are those presenting symptoms of general unthriftiness, indigestion, intractable diarrhœa, ulcerated mouth, or such like. Had these cases been early put under proper supervision, it is quite probable that the symptoms complained of would not have arisen.

As I believe that many dogs do not show any further symptoms of the disease than the skin eruption, so I say that if such cases are isolated and given such care as to ensure good nourishment and to

prevent chill or secondary infection, they will, more often than not run a mild course and make a good recovery without further treatment. The course of the disease, however, must necessarily depend upon the virulence and source of infection. Granted the early recognition of the disease, treatment is of the simplest, and frequently no medicine is required.

When a case is not seen early, or when a simple case has become secondarily infected, or infected from a fresh source, or subjected to chill or other lowering influence, such as the abuse of drugs, or when a relapse has occurred, then very often great difficulty is experienced in bringing such a case to a satisfactory termination. Here good nursing is of paramount importance, and here also skill is required in the choice of appropriate drugs suitable to the case in hand. Naturally a wide discretion must be allowed to the medical attendant in his choice of drugs, and each case must be treated on its merits, and so-called "routine practice" (which, to my mind, is another name for professional laziness) must be abolished. Drugs which are suitable to many cases in one outbreak may be useless to those of another. On many occasions more good may be done by refraining from dosing than by prescribing medicine. This should be made clear to the owner, that he may not think his adviser lacking in ability to treat the case, and so lose confidence because there is no syrupus rosæ in the dispensary. Though it may go against our financial interests, occasionally, at least, our business instincts must be made subservient to our professional training and dignity.

In an excellent chapter devoted to the treatment of distemper in a recent book on therapeutics a well-known writer gives one or two prescriptions for almost every symptom of the disease that may be presented. This is typical of most contributions to our literature on the subject, and I regret it because such essays often make a profound impression upon the reader, especially if he be a student, and tend towards making him blindly follow the lines laid down. His method becomes stereotyped and himself unable to take a broad view; in fact, he drops easily into routine practice. Anyone reading such a work is led to believe that medicine must play a great part in treating distemper, and that each symptom must be treated as it arises, each with its own bottle of mixture. This may be good for the pocket, but I do not believe that it is good for the patient. It is hard to say that each symptom is benefited by the medicine prescribed for it, for other similar cases get well just as quickly, and often more quickly, when no special medicine has been given. For instance, I think it a mis-

take to try to stop the intermittent diarrhœa. Untreated, it often alternates with obstinate constipation, and the treatment of the diarrhœa will probably only aggravate the constipation, and *vice versa*.

In any case it is most necessary that the administration of medicine shall not in any way interfere with the feeding of the dog. It is of greater importance that he should feed voluntarily than that he should be nauseated with castor oil and buckthorn or put off his food with opium, paregoric, sweet spirit of nitre, or such like.

This brings me to the subject of nursing. To my mind, the sooner the patient on being recognized as suffering from distemper is isolated as a sick dog the better his chance of recovery. He should be rigidly kept from immediate or intermediate contact with all other dogs for at least a fortnight. The other general conditions most necessary are plenty of fresh air and sunlight if possible, careful protection against damp and all depressing influences, together with the regular administration of nourishing food. To such a meeting as this I need not insist on scrupulous cleanliness. Unless complications have arisen warmth is not very essential, though very low temperatures and rapid variations should be avoided. From 50° to 60° F. is a good temperature. Dogs do badly in hot, stuffy rooms. The best food is undoubtedly milk. I think it is best to put a patient at once on a milk diet, allowing him as much as he will take to drink, and feeding him with rice puddings or sponge cake soaked in milk, and such-like foods. If milk in quantity makes him sick, it may be tried diluted with Vichy water, which dogs take readily. Lactol is useful, but I have had best results from feeding on Savory and Moore's prepared food, especially in puppies. Meat juices may be used provided they are not too salt. Often they are very gelatinous, and this, while making them appear dainty, frequently causes undue thirst. The best meat juice, in my opinion, is that which we can prepare ourselves by pressing fresh, lean, raw beef.

Food, of whatever kind, should be given in small quantities and frequently—that is, *at least* four times in twenty-four hours. If the case becomes complicated, and even when recovery seems assured, the greatest care must be taken over this question of feeding. No matter how well fed the patient, he will lose flesh and condition; till the infection has, so to speak, exhausted itself, no amount of nourishment will cause him to gain weight. This rapid loss of flesh often leads the owner to believe that the dog is not getting enough nourishment, and consequently the patient is forced with more food than his system can assimilate. This is almost worse than starvation, and is

liable to cause severe complications, such as diarrhœa, vomiting, or convulsions. What little the patient will take voluntarily is of infinitely more value to him than the best of nourishment forced upon an unwilling stomach.

Particular care should be taken to see that any sudden ravenous hunger following a poor appetite should not be gratified. This always denotes some critical change in the course of the disease, and frequently, if it does not usher in convulsions, these at least are almost certain to follow a good meal after a period of anorexia. For a similar reason a sudden change to solid food is decidedly dangerous. If an uncomplicated case has run for a fortnight, and the patient seems to be going on well, he may be taken out in the open air during the warm hours of the day if the weather be good, but he must not be urged to exercise himself, and he must not be allowed to get tired. Violent exercise should be strictly prohibited for another fourteen days at least. A long walk or a quick run, or even a half day with the guns for a sporting dog may cause a serious relapse, and this is far more difficult to treat than the original illness.

Common-sense should guide us in these as in all things, and we should not expect more of the dog than we should of ourselves had we been suffering from a disease of equal severity.

As to the treatment of the disease by the aid of drugs I have not a great deal to say. No drug seems to be of undoubted use in all cases, but there are a few which are of especial value in a large number. Quinine and the salicylates are used by nearly all in some form or another, and more often than not with some good results. For very many years yeast has been used and with very good effect. At the present day I believe it the most valuable remedy we possess. I have tried various preparations of it, and I think the best is nuclein or nucleinic acid. If started early in the disease and continued steadily it has a marked beneficial effect. It seems to assist the tissues to resist the infective process. Another general remedy which may be used with success, both as a food and as a curative agent, is sour milk. Some dogs will drink this readily, and provided they will drink sufficient it is remarkable how well they do on it. Small doses of thyroid extract also seem to have a beneficial effect on tissue metabolism and help the patient to throw off the disease. I assume that this is so as there is nearly always some steady increase in weight after its administration.

To review all the many and various phases of complicated cases of distemper and to give in detail my ideas as to the best method of

treating them would occupy too much of your time and, I fear, would prove tedious. Let me therefore merely call to your minds some of the difficulties which we have to contend with in treating cases, and state shortly what I believe is best to be done.

The greatest difficulty is generally loss of appetite. This is a matter more for the nurse to contend with than the medical attendant, but the latter must have a list of palatable and nourishing foods at his finger ends, so that he may be able to suggest a suitable diet. Some dogs prefer dry food, and such dogs generally prefer lean meat cooked or raw. Sometimes they will not eat meat, but they will often readily eat stale sponge cake or sweet biscuits, if given dry. If the patient will take sloppy foods, stewed rabbit and rice will often tempt him when all else fails. Chicken and game are of little value, and only too often are not digested. If food has to be forced upon the dog fresh raw beef juice or milk are the best foods that can be chosen.

If there is no lack of appetite the disease generally terminates favourably, especially if the dog does not lose flesh to any great extent. With regard to the diarrhœa, which is so often troublesome and intractable, this does not seem to need treatment by drugs, as it will nearly always stop of itself, though it is often the last symptom to disappear. The trouble arises in the fact that it frequently is the precursor of dysentery and mucous colitis or ulceration of the rectum —conditions which may prove impossible to cure. The bismuth salts are useful here, as are also hæmatoxylin and opium. I have found rectal injections not of great benefit, and their administration is generally objectionable, both to the patient and his owner. Some few cases have recovered wonderfully by the use of chlorodyne, but I find the opiates as a rule very uncertain in their action on the bowel of the dog. Milk is the best diet, fresh or peptonized, and, if the dog will take it, sour milk will often effect a cure when ordinary medicines fail. If the case runs on to dysentery or ulceration of the rectum, it is nearly always fatal, and there is little that can be done. The incessant tenesmus is almost impossible to stop, though the opiates have some beneficial effect. In some of these cases, when I have had the opportunity of making a *post mortem*, I have found the cæcum and upper part of the posterior bowel so enormously dilated as to occupy three parts of the abdominal cavity. This over-distension will, of itself, cause continual straining, and in such cases I know of no cure. The next greatest difficulty, in my opinion, is the satisfactory treatment of the nervous symptoms that are so common in the disease. The most fatal are undoubtedly meningitis and coma. The convulsions and the general

attitude and manners of the patient are very distinctive in meningitis, and such cases are best destroyed as soon as possible. Setoning in the nape of the neck has been advocated, but I have not found it of any use, and I think the benefit to be derived from such a kind of treatment would have to be very sure and certain before I should advise such a very cruel expedient.

Coma is nearly as unsatisfactory to treat. If stimulants are given the probability is that coma will only give place to delirium and death. Ordinary convulsions are not so fatal, and many cases are saved by prompt treatment. It is most important to take action in time, before the fits are frequent or severe, and especially so if they are associated with high temperature. While they only consist of a champing of the jaw, or abnormal dribbling of saliva, they may be stopped by the use of bromides. I find strontium bromide in syrup most effectual, and the drug may be pushed to such a point that the dog loses all power in his limbs. After three or four days, as the drug is discontinued, the muscles regain their function and the fits do not recur. In meningitis bromides are no use at all, and if anything they only irritate and make the case worse. In chorea, when the disease is established or become chronic, I do not believe that drugs are of any value. If a case is going to get well it will get well without their use. In the early stages, however, when the temperature is high, aspirin, antifebrin, and such-like drugs are very useful, and it is always my practice to make use of them, for I believe that in this way the progress of the disease may be checked.

Jaundice is believed generally to be one of the most fatal symptoms of distemper, and most difficult to treat. I cannot say that this has been my experience even among cases in several packs of hounds. I have generally found that the patients die of catarrhal pneumonia, which only too often supervenes. If the patient can be protected from lung complications the jaundice will generally give way to treatment, but the treatment in this case must be very active, and milk is the only safe diet. I have had bad cases even in such breeds as bloodhounds, setters, and Japanese, and the losses have only been among those in which there were lung complications. In this, as in all other phases of distemper, it is essential to see one's patient early. Probably the greater part of the mischief is done when once the yellow colour becomes apparent. The greatest difficulty experienced is to get the bowels to act freely and to get the owner to give the case sufficient nursing and sufficient food.

The last difficulty I shall mention is one which I very frequently

have to meet. I refer to the fact that breeders refuse to admit that dogs in their kennel are really suffering from *distemper*. You may call it by any other name. Influenza is, perhaps, the most popular.

It is most difficult to make the public realize that a dog with the characteristic eruption is already infected with distemper. They do not want the dog to have it, and, so long as the animal is not visibly ill to them, they will not believe, and they will not take the necessary precautions. Veterinary surgeons can act together and make it a point of honour to reject all such dogs from shows. This united action would sadly deplete the benches and greatly chagrin show committees, but it would go a long way towards decreasing the at present terribly high mortality of the disease. As I have repeatedly pointed out, the present veterinary examination at dog-shows is an absolute farce, and it is lowering to us as a profession; and I, for one, shall resolutely refuse to act for any show under the present unsatisfactory conditions.

Let me now sum up my remarks, in conclusion.

It is very necessary to recognize the disease in its earliest stages, and take the case in hand for treatment. Nursing must be of the very best, and medicinal treatment of the very simplest.

There must be no excess of nourishment, but what is given must be of the simplest and most easily digested, and, if possible, taken voluntarily.

The greatest care must be taken to prevent fresh infection and relapse.

Remember the severity of the disease, and take every precaution to ensure a good convalescence. Remember how long it takes a human being to throw off influenza and such-like diseases, and do not expect the poor dog to do much better than the more highly developed creature—his master.

NOTES ON *TRYPANOSOMA DIMORPHON*—CORRECTIONS.

In the Veterinary Journal for January of this year we published an article by L. E. W. Bevan, M.R.C.V.S., under the heading "Notes Concerning *Trypanosoma Dimorphon*." We have received the following corrections from Mr. Bevan: The last paragraph on page 13 commencing "As is well known . . ." to "extension of the organism first found in the Gambia," on page 14, should have been in inverted commas, being a quotation from Montgomery.

Mr. Bevan also writes: "Under measurements (page 16) I should have said 'forms about 20 μ long predominate' . . . not '30 μ.'"

THE DRUG TREATMENT OF CANINE PIROPLASMOSIS.[1]

By G. A. F. NUTTALL, M.D., Ph.D., Sc.D., F.R.S.
Quick Professor of Biology, Cambridge.

The Continued Infectivity of the Blood of Dogs which have Recovered from Piroplasmosis after Treatment with Trypanblue and Trypanred.

In a previous paper by Nuttall and Hadwen the cases of four dogs were described which had survived an attack of piroplasmosis consequent upon treatment. The dogs (Nos. 1, 3, 4, and 6) appeared quite well at the time we published the account of our experiments. Whereas some of our other dogs which had been successfully treated succumbed to distemper, the four referred to escaped this intercurrent disease or (Dog 6) recovered from it. The following data refer to these four dogs, whose previous history is in each case briefly summarized, so as to render the subsequent record more intelligible.

Except for Dog 4, which is showing increasing signs of advancing age, all of the animals are at present healthy as far as outward appearances are concerned.[2] The body temperature has remained normal in the cases of Dogs 1 and 3, and in the cases of Dogs 4 and 6 has shown occasional slight rises which may have been due to other causes. The body weight has increased in all cases: from 15 to 17lb. (232nd day, Dog 1), from 17½ to 21lb. (235th day, Dog 3), from 20 to 25lb. (227th day, Old Dog 4), and from 16 to 20lb. (210th day, Dog 6). Parasites have not again been detected in the peripheral blood upon microscopic examination.

On the other hand, the parasites must have persisted in the animals, for blood taken from the ear-veins of the four dogs gave rise to fatal piroplasmosis in all of the test-dogs which were inoculated with it. Thus:—

The blood of Treated Dog No.	Day after inoculation of Treated Dog when its blood was collected	No. of Test Dog inoculated	No. of days after inoculation when Test Dog died of piroplasmosis
1	143	I.	19
3	190	II.	16
4	182	III.	13
6	165	IV.	21
6	210	V.	34

[1] This article is abstracted from a very excellent paper on the subject, a reprint of which Professor Nuttall very kindly sent us. The original paper appeared in full in *Parasitology*, vol. ii., No. 4, February, 1910, and contains, in addition to our abstract, a *résumé* of the earlier work by various Italian investigators, and the details of experiments by Nuttall with the aniline dyes, brilliant green, benzopurpurine, and Congo red. Previous articles on the subject have appeared in the Veterinary Journal of December, 1909, and January, 1910.

[2] Dog 6 has died since this was written. It was killed by fighting with another dog on the 236th day after inoculation.

It is obvious from the foregoing that the treated dogs behave, in respect to the survival of the parasites in their blood, similarly to animals which recover under natural conditions. They represent "salted" animals in which parasites persist for an indefinite time without apparently producing any ill effects, the number of parasites present in the blood being exceedingly small. Although the test dogs which were inoculated with the blood of the recovered animals lived somewhat longer than is usual after inoculation with the blood from acute cases of piroplasmosis, there is no evidence that the parasites in the blood of the recovered animals had in any way become attenuated.

Two of the recovered dogs were, moreover, re-inoculated with virulent blood, but neither of them showed any reaction. Dog 3 was re-inoculated on the 221st day after the first inoculation, and Dog 4 was similarly re-inoculated on the 213th day.

Conclusions.—It is evident from these experiments, protocols concerning which are given in the original paper, that the parasites persist in the blood of dogs which have recovered from an acute attack of piroplasmosis in consequence of treatment with trypanblue and trypanred. The blood of such dogs was found to be infective and virulent 143 to 210 days after the treated dogs were first inoculated. In respect to the persistence of the parasites in their blood, the successfully treated dogs behave similarly to dogs which have recovered naturally. Two of the dogs which recovered after treatment were re-inoculated, with negative results, on the 213th and 221st day after the primary inoculation which produced the acute attack from which they recovered.

Summary and Conclusions.

The following summary and conclusions apply to the results of the experimental treatment of canine piroplasmosis by various authors, including myself:—

(1) Drugs which have been found ineffective in the treatment of canine piroplasmosis are:—

Arsacetin tested on one dog by Nuttall and Hadwen (1909).

Soamin tested on one dog by Nuttall and Hadwen (1909).

Sodium-methylarsenate tested on two dogs by Nuttall and Graham-Smith (1908).

Tartar emetic tested on two dogs by Nuttall and Graham-Smith (1908).

Sodium nucleinate tested on two dogs by Levi della Vida (1907).

Beta-naphthylamine tested on two dogs by Nuttall and Graham-Smith (1908).

Brilliant green tested on two dogs by Levi della Vida (1907).

—— tested on one dog by Nuttall (1909).

Benzopurpurine tested on one dog by Nuttall (1909).

Tryparosan tested on two dogs by Nuttall and Hadwen (1909).

Methylene blue tested on two dogs by Levi della Vida (1907).

—— tested on two dogs by Nuttall and Graham-Smith (1908).

(2) Of doubtful value are :—

Fowler's solution tested on one dog by Levi della Vida (1907). The dog is stated to have had but a mild attack of piroplasmosis.

Atoxyl tested on six dogs by Gonder (1907) with negative results in that all of the dogs died of piroplasmosis. Levi della Vida (1907) states that he cured some dogs by atoxyl treatment, but does not state how many.

Quinine bihydrochloride tested on one dog by Nuttall and Graham-Smith (1908) with negative result. Memmo, Martoglio, and Adani (1905) state that they cured two dogs by injections of quinine, but give no particulars of their experiments.

Oil of turpentine tested on three dogs by Levi della Vida (1907) with negative results. Memmo, Martoglio, and Adani (1905), however, claim to have cured all of the twelve dogs they treated with turpentine.

(3) Apparently the following drugs may exert some effect :—

Dichlorobenzidine + two molecules of amido-napthol-disulpho, tested on twelve dogs, two of which recovered, Levi della Vida (1907).

Sodium cacodylate tested on seven dogs, six of which are stated to have recovered, Levi della Vida (1907). (I am about to test this remedy afresh, since the Italian author gives very few particulars regarding his experiments).

Congo Red tested on three dogs by Nuttall (1910). All of the dogs died, but the drugs exerted a decided effect upon the parasites.

(4) Definite curative or preventive effects are exerted by :—

Trypanblue ($C_{34}H_{24}N_6O_{14}S_4Na_4$, the tetrazo compound of toluidine and amidonaptholsulphonate of sodium) cured seven out of nine dogs in the experiments by Nuttall and Hadwen (1909), and when given early prevented the appearance of the parasites in the dogs' blood. These results have been confirmed by Jowett (1909) who tested the dye on fourteen dogs, two of which were moribund. The latter died, whilst twelve

recovered. Six of Jowett's cases were due to experimental infection, the others were cases of naturally acquired infection, and a number showed severe clinical symptoms. The drug is ineffective when given by the mouth.

Trypanred tested on two dogs by Nuttall and Hadwen (1909) caused the parasites to degenerate and disappear. (Further experiments with this dye are in progress. The initial experiments indicated that trypanred is not as satisfactory as trypanblue for purposes of treatment as it produces a more irritant effect.)

(5) In dogs which have recovered from piroplasmosis after treatment with trypanblue and trypanred the blood remains infective and virulent for six to seven months or more after treatment. Such dogs resist reinoculation with virulent blood obtained from dogs suffering from acute piroplasmosis, and consequently are to be regarded as "salted" animals. From a practical point of view the "salting" of dogs (and cattle) by the method of inoculation followed by the dye treatment appears to offer decided advantages. The method is at present on its trial in respect to cattle which are being "salted" in England prior to their exportation to Africa.

(6) In judging of the value of dyes in the treatment of piroplasmosis it is essential that accurate observations should be made with regard to their influence upon the parasites as well as upon the symptoms of the disease. Both in dogs and cattle the injection of trypanblue causes the pyriform parasites to disappear rapidly, the remaining parasites appear rounded and degenerated, and ultimately disappear from microscopic observation. Although the parasites may reappear in small numbers after some days their presence is not accompanied by outward symptoms, and the animals proceed to recovery. Relapses may, however, occasionally occur with the reappearance of parasites. Fatal relapses have only been observed in two of our dogs. The influence of the treatment upon the fever is very marked. Both in dogs and cattle suffering from piroplasmosis, the temperature usually falls rapidly to normal, and coincident with the fall in temperature there is a general improvement in the appearance of the animals. The effect of the dye appears to be to cut short the acute attack, and to convert it into a mild, chronic form of the disease, which seems to exert no injurious influence upon the animals. In fact, the animals may rapidly improve in condition as evidenced by a gain in body-weight and in appearance. Having recovered from the acute attack the animals harbour the parasites

in small numbers for a long time, and by virtue of the continued presence in their bodies of the piroplasms they continue, for a like period of time, to remain resistant to reinfection. Whereas the animals cannot be described as "cured" in the sense that they are rendered free from parasites, they are rendered proof against reinfection; in other words, they are "salted."

Judging from the immediate effect of the dye upon the parasites and upon the symptoms of piroplasmosis both in dogs and cattle, and the subsequent behaviour of the animals after treatment, I am fully convinced that trypanblue will prove of great value in practice until such time as we shall discover an equally efficient remedy not possessing the colouring properties of the dye. The fact that trypanblue dyes the tissues for a considerable length of time will necessarily militate to some extent against its usage in cases where cattle are intended for meat consumption, but it will assuredly protect valuable breeding animals which it is proposed to introduce into a country where piroplasmosis prevails. In the case of dogs, this objection will scarcely weigh in the balance as against saving the animal from a disease which is so commonly fatal. That the coloration of the tissues disappears with time is clear from the case of Dog 6, which was killed on the 236th day after the dye was injected, and found to be quite normal in appearance. Only further observations can determine how long the coloration of the tissues lasts in cattle and dogs, and it would be well if different observers recorded their experiences in this particular.

At present there exists to my knowledge no other remedy which will cure piroplasmosis in either cattle or dogs.

Clinical Articles.

THE VALUE OF RECTAL EXAMINATION IN CATTLE.

By AINSWORTH WILSON, F.R.C.V.S.
Witham, Essex.

It will be generally admitted that the palpation of the internal genital organs *per rectum* is often indicated in the larger domestic animals; a proper diagnosis of the various abnormalities and pathological conditions, which may or may not lead to sterility, can be made with certainty in a large proportion of cases.

A positive diagnosis may be of the greatest importance to determine the existence of pregnancy at different stages of gestation; internal examination is often necessary for this purpose.

The question may be asked, "Do we, as a profession, attach sufficient importance to rectal examination in cattle practice?" Personally, I have long felt that it has not received quite the attention it deserves in daily practice.

Many of us, I take it, can call to mind cases in which rectal exploration might have proved most useful, but unfortunately it was not resorted to.

This neglect, if neglect there be, does not apply to horse practice, in which the value of internal examination is universally recognized, more especially in bowel cases; and there are few practitioners who do not, as occasion arises, manipulate the pelvic and abdominal viscera through the rectal walls.

The two papers on Sterility recently published in the Veterinary Journal suggest the need for more extended and systematic exploration *per rectum et per vaginam.*

The editors are to be congratulated on bringing the valuable experiences of Continental experts within the reach of country veterinarians, to many of whom the original papers presented at The Hague last autumn must remain a sealed book.

Those who aspire to conquer sterility in bovines, and who make a systematic examination of the sexual organs, know how complex and exacting the problem really is. It requires the expenditure of a good deal of time and trouble, together with the keeping of careful records of each individual case.

To begin with, much practice is needed in the palpation of the genitals of the healthy cow at various physiological periods; while both the non-pregnant and the pregnant animal demand attention.

The experience and confidence gained in this way are of the greatest service in the detection of disease of any part of the genital tract, *e.g.*, ovarian cysts or persistent corpora lutea, or both; thickening or occlusion of the oviducts; chronic catarrhal or purulent endometritis; pyometra and other collections in, and diseases of, the uterus; mummified or macerated fœtus; imperforate vagina; suspected retention of fragment of after-birth with contracted os; torsion of uterus with occlusion of vagina; spurious pregnancy; straining, uneasiness, or colicky pains during gestation.

In the male animal the accessory glands near the bladder and a portion of the vasa deferentia are easily distinguished, and sexual soundness, so far as they are concerned, can be ascertained. The above will suffice to show the opportunities for manipulative or operative handling by the rectum in both mare and cow.

Rectal manipulation is not, however, confined to the organs of reproduction. It is most useful, in fact indispensable, in palpating the pelvic and some of the abdominal viscera, together with the bones, ligaments, glands, and blood-vessels. The bony and ligamentous framework of the pelvis and a considerable portion of the sublumbar region can be examined with more or less facility for fractures and other lesions. In paraplegia, for example, rectal examination should never be omitted. Cysts, abscesses, tumours, or dropsical collections may also be detected occasionally.

The urinary organs are well within reach, except the kidneys, which in big cows are situated too far forward for effective manipulation. The alimentary tract presents portions of the rumen, together with the small and large intestines. Several posterior groups of lymphatic glands are also within reach, a fact of some importance in the diagnosis of abdominal tuberculosis. Other pathological conditions of the tissues and organs available for manipulation will readily suggest themselves.

The following notes may help to emphasise the value of exploration *per rectum* in affections of the genital apparatus:—

I.—Pelvic Constriction—Artificial Abortion.

The subject was a nymphomaniac cow, aged 6. She had her last calf twenty months before. She became pregnant after sixth operation for cystic degeneration of (1) right ovary; (2) both ovaries. I examined her December 31, 1909, and found deformed pelvis with fractured shaft of ilium, and large callus formation. Normal parturition would have been impossible. The right ovary was the size of

a pigeon's egg, fibrous; left ovary, walnut size and little nodulated. The left horn was impregnated and the os closed; mucus thick and normal, and sacro-sciatic ligaments slightly relaxed.

Examined January 2, 1910, sixty hours after operation. The cow aborted a fresh four months calf. Two weeks later she showed signs of œstrum, and thereafter came on every three weeks. Neither the cysts nor the nymphomania recurred while she was being fed for the butcher.

II.—Mummified Fœtus—Dumb Œstrum.

The subject was a shorthorn cow, that had been clinically tuberculous two years. She had her sixth calf in December, 1908, was bulled in March, 1909, and believed by the owner to be pregnant.

I examined *per rectum* on first appearance of œstrum two to three weeks before the date she was due to calve. The left cornu was normal, but a mummified fœtus was in the other, 2 to 3 in. thick, 6 to 8 in. long, a firm, semicircular, irregular body; some bones crushed like egg-shells, curved spine plainly distinguished. The os admitted the index finger; ovaries normal, œstrum recurred normally and regularly, and the fœtus remained *in situ* for many weeks while the cow was being fattened; the os was closed between the periods.

III.—Spurious Pregnancy—Myxometra.

Jersey heifer, aged 2, believed in calf, was brought in from grass, and examined August 13, 1909. She had a poor appetite, upper flanks hollow, abdomen rather distended, eyes little sunken, heart's action rather weak, constipation and grinding of the teeth, temperature 102·6° F. There was a little reddish slime on the roof of tail and ventral surface; vulva very small (admits two fingers). I administered a draught containing ol. lini., pulv. opii and calomel; and left sodii bicarb.

August 14, 1909: Small quantity red mucus was found *per vaginam*. On examining *per rectum*, the uterus was found to be large, fluctuating, and more or less shapeless; sac containing several gallons fluid, cervical canal probably open. I attempted unsuccessfully to turn uterus and reach ovaries. I then administered hydrastis canadensis, and left large doses sodii bicarb. There was no straining, and the general health was improving.

August 15: Further improvement, but more discharge. The right ovary was reached with difficulty, and two yellow bodies pressed out. The left ovary was beyond reach under the distended horn.

August 16: The vulva had been relaxed, and admitted left hand; the cervix was further dilated with the fingers.

On the 18th the floor of box flooded with a slimy, odourless, red-tinged fluid, and the uterus was small on exploration. Involution proceeded rapidly and œstrum appeared nine days later. After three weeks' period she went to bull and proved in calf.

It is probable that this heifer aborted very early in pregnancy. This would account for the fluid in the uterus. She was one of nine, eight of which were forward in calf.

TREATMENT OF SUPRA-SCAPULA PARALYSIS.

By Professor W. C. SCHIMMEL.

Director (Principal) of the State Veterinary School, Utrecht.

In consequence of the case of supra-scapula paralysis communicated in the Veterinary Journal of March, 1910, by Captain G. P. Knott, A.V.C., may I be allowed to remark that this form of lameness, as a rule, cures in a short time, if only rest is given to the horse for at least a fortnight after the beginning, and when afterwards it is obliged to exercise itself daily in increasing measure. It is possible that an extravasation of blood in the course of the supra-scapula nerve has caused the paralysis by a compression of the nerve. Its resorption is favoured by rest. However, ten to fourteen days having passed, the horse, before the supra- and infra-spinatus muscles become wasted, should be exercised twice daily for at least half an hour, however defective the gait may be.

As Captain Knott observed, too, it can regularly be seen that the lameness diminishes proportionately to the increasing motion, and that in the same way the atrophy of the scapula muscles is respectively prevented or improved.

In most cases the horse begins too late with the forced and methodical continued motion; this happened *in casu*, too, and therefore it lasted such a long time before recovery was attained. Any other treatment of this paralysis is almost in vain; with massage, blisters, electricity, subcutaneous injection of veratrin, strychnine, &c., usually no cure is effected if the horse is kept at rest.

Walking exercise in the beginning, soon followed by trotting exercise, is the remedy by which recovery of the paralysis is generally brought about in a short time. This knowledge makes the prognosis of the fresh cases of supra-scapula paralysis as a rule favourable.

DISPLACEMENT OF THE URINARY BLADDER IN A HEIFER.

By S. J. MOTTON, M.R.C.V.S.

Penzance.

The term prolapse when applied to the urinary bladder designates a condition in which the bladder passes through a rupture of the vaginal wall so as to rest in the vulva or even project beyond the vulva. Except as such a prolapse, it would on first consideration appear to be impossible for the uninverted bladder, or any part of it, to protrude from the vulva. Such an accident, however, may occur, and the following case illustrates the fact.

On March 7 last, after difficult parturition, a small Guernsey heifer was observed to be down, unable to rise, and straining violently at intervals. Inability to rise after difficult calving is not very rare, and the owner, feeling satisfied that this would pass off in a few days, did not concern himself about it.

The afterbirth was expelled soon after the calf, and the attendant was at a loss to explain the straining until he noticed a rose-red prominence peeping from the vulval labiæ. However, like many of his kind he trusted to luck, and it was not until March 12, five days after it was first noticed, that we were sent for. The animal was still down and unable to rise, but she maintained the correct recumbent position, could turn herself, was feeding, chewing her cud, and had a normal temperature. The mass protruding from the vulva had now much increased in bulk, and outside the vulva had attained about half the size of a regulation Association football. Its surface was covered by the mucous membrane of the vagina, which was abraded in places.

So tightly did the protrusion fill the entrance to the vulva that considerable difficulty was experienced in passing a hand into the vulva. The os uteri was situated above it, and on palpation there was no room to doubt but that it was the urinary bladder displaced into the vulva and much distended by urine.

The wall of the vagina was, except for the above-mentioned abrasions, quite intact, but was, of course, very much stretched. The bladder had in some way been forced out of its normal position during parturition.

Before the bladder could be returned to its proper position it was necessary to remove the urine. This was accomplished by opening the urethra (which, like the bladder, had turned on itself)

with a finger, and owing to the sharp bend present in the urethra, this was at first difficult, but necessarily became more easy as the bladder decreased in size. But even when empty the organ could not at once be kept in its correct position because of the straining, which was now, however, less violent.

In order to prevent the bladder from coming outside again the vulva was stitched. A few doses of sedative medicine were prescribed, and the vagina was washed out daily with a warm antiseptic solution. During the four succeeding days the catheter was passed once daily. At the end of that time the bladder had recovered sufficient tone to empty itself, and was now occupying almost its normal position, while the wall of the vagina had considerably contracted. The heifer was strong enough to stand for an hour or two daily. To-day (March 28) she is well enough to be turned out to graze daily, and there appears to be no reason to fear any further trouble.

SINUS IN THE INFERIOR MAXILLARY BONE.

By E. WALLIS HOARE, F.R.C.V.S.

Cork.

Subject.—An aged farm-horse.

History.—The owner had for some time observed that the horse had difficulty in feeding; a swelling appeared on the lower jaw on the near side, which gradually increased in size.

On examination, the lower jaw was enormously swollen, hard to the touch, and painful. On the lower aspect, a short distance anterior to the submaxillary artery, an opening was discovered which admitted a probe. The instrument could be made to enter a large cavity formed in the bone.

The horse was cast, and an examination of the teeth carried out. The second molar tooth on the affected side was missing and a probe could be passed from the external opening in the jaw into the alveolar cavity of the missing tooth.

On manipulating the probe, a loose body could be distinctly felt in the cavity formed in the bone. This of course had to be removed, but the difficulty was to enlarge the opening so as to permit extraction. I tried a small trephine, but, in consequence of the thickness and hardness of the bone, it would not work. So I got a saw with a very fine point (one used for cutting key-holes), and after much

difficulty succeeded in enlarging the opening in the bone and removing the foreign body, which proved to be a molar tooth, much decayed and black in colour.

I next made a communication between the opening in the bone and the alveolar cavity, so that irrigation could be carried out. The cavity in the bone contained a very large amount of semi-masticated ingesta, which was removed after some trouble.

I regret that I cannot give the after-history of the case, as the animal was taken home next day, a journey of 30 miles; but I conclude that he must have recovered, as the fee was paid and I have heard nothing since from the owner.

If the fee had not been paid I should certainly have heard of the result: most probably it would not have been favourable.

SILVER WIRE SUTURES.

By W. STAPLEY, M.D., M.R.C.V.S.

Professor of Veterinary Surgery in the University of Melbourne.

HUMAN surgeons do not as a general practice bury silver wire. I have done so because I have been unable to close my abdominal wounds without tension too great for the practical application of chromic gut. I prefer silver wire to kangaroo tendon and to silk. Silk is not absorbed and as a permanent suture it is more liable to create trouble than wire. My experience of tendon has been unsatisfactory. I have had abscess follow its use. It is slippery stuff to work in a tense abdominal wall. Of course, surgeons teach that stitches should not be put in under tension. Had we followed this law our cases would have had to remain unsutured!

As yet, we have no aluminium bronze wire in Melbourne, so we are unable to use it. Economy suggests its use in place of silver wire. Copper wire electro-plated is difficult to use because the copper is exposed at the cut ends.

Canine Clinical Notes.

AN INTERESTING CASE OF CHOKING IN A DOG, WITH AN UNCOMMON SEQUEL.

By G. H. LIVESEY, M.R.C.V.S.

Hove.

The subject of this note was a dachshund which met his death as as the result of swallowing a sharp bone which became arrested in the thoracic portion of his œsophagus and eventually penetrated his aorta.

I was hastily summoned to the dog very early one morning because he was vomiting blood. When I arrived at the house I was informed that the dog had jumped off his mistress's bed, on which he had been lying, and had three times vomited a considerable quantity of blood. On examining the vomit I found that it was practically pure blood, now coagulated, and not blood-tinged mucus. I then thought he must have ruptured a blood-vessel in his stomach.

The owner informed me that the dog had been under treatment at Lewes by Mr. Munby for subacute gastritis for five or six days previously. The only other history obtainable was that he had been into a wood on a foraging expedition, and on his return he was sick, as though he had been eating filth. Since then he had been difficult to feed, and had frequently vomited.

I subsequently consulted with Mr. Munby over the case, but the owner was so distressed by the frequent vomiting of blood that she persuaded me to take the dog home with me later in the day. Early the next morning, however, we found him dead, after having vomited a huge amount of blood.

The *post-mortem* examination revealed a piece of bone (vertebra of a sheep) lodged in the œsophagus just behind the level of the second rib. The transverse process of the offending lumbar vertebra had been broken and was wedge-shaped, and the point had been driven through the œsophageal wall and into the common aorta. When the bone was *in situ* its wedge-shape quite closed the aortic wound, but when the dog moved about, and the bone was slightly displaced, blood evidently passed along the track and into the stomach. When the bone was removed a probe could be passed easily through the œsophageal and aortic wounds into the heart. It is remarkable that the tough elastic tissue of aorta could be pierced in this way. No doubt the puncture was completed when the patient jumped from the bed, as no blood had been vomited until then. The ulceration of the œsophagus indicated that the bone must have been there quite a week.

GANGRENOUS STOMATITIS AND GASTRITIS IN A DOG —SPONTANEOUS RECOVERY.

By GEO. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.

Professor in the Royal Veterinary College, London.

This most interesting case came under my notice on March 24. The patient was a mongrel Airedale terrier, male, about 2 years old. He followed the owner, a most intelligent boy, about 14 or 15 years old, into the house six weeks previously and refused to be sent away, so the boy adopted him. For the first three weeks the dog appeared perfectly well, but then he commenced to vomit. His appetite remained fair. He would eat small quantities of food, consisting of flesh meat, and biscuits, sometimes dry and sometimes soaked, and as he never took much at a time he was able to retain it. Frequently, however, during the day he would vomit a blackish liquid, very offensive in odour. He next began to dribble "black slimy stuff." Then diarrhœa commenced, the fæces being soft and yellow. The owner thought that the illness was probably due to worms, and so "worm medicine" (areca nut) was administered, but no worms were seen either before or after the dose. A week before being brought in to the clinique, while the dog was being fed on some fresh red beef he spat out "a dirty yellow bit of flesh, thin and doubled up." Since then he had fed better and got brighter in manner. The boy was not satisfied with him, however, because he remained thin and behaved in a peculiar manner when given water, appearing to "bite at it instead of lapping it." The above is the clear and lucid history as given by the boy, some of his own words being in inverted commas.

On examining the dog I found him to be quite bright, but thin and rather weak, in fact, when he jumped from a comparatively low table his legs collapsed under him, but he immediately regained his feet. There was no discharge from his eyes, and the colour of his conjunctiva was normal. On opening his mouth the cause of his inability to lap water was immediately visible. He had lost the whole of the fore portion of his tongue right back to the *fraenum linguæ*. No doubt this was the "dirty yellow bit of flesh" of which the boy had spoken. The wound of the stump of the tongue looked to be healing nicely, and there was now no offensive odour. The dog has not vomited for almost a week, and his appetite is improving. His temperature was 102·2° F. I offered the dog water, and the boy's description of the dog's method of trying to drink was shown to be quite correct. It took him a very long time to get even a small amount, and, of course, he got it more easily from a deep basin than from a shallow one.

I prescribed a little tonic medicine and a mild collutorium, and gave instructions as to feeding and providing water in a deep basin. The dog is now thriving well (March 31), and is learning to suck water. He is much stronger, and is rapidly gaining flesh, and the wound of the tongue is quite healed.

I am prompted to record this case to show how spontaneous recovery may occur in some cases of very serious disease. This dog was affected with a disease closely simulating the so-called "Stüttgart dog disease," which is undoubtedly fatal in a very large proportion of cases where the necrotic mouth lesion occurs. This dog, at any rate, had not had his natural recuperative powers reduced by the administration of nauseous drugs. Of course it must be remembered also that the patient was a young dog (estimated at 2 years old) and a mongrel, both points which, in my experience of this disease, assuming it to have Stüttgart disease, were enormously in his favour. On the other hand, I do not wish to detract from the beneficial effects that may be obtained from the judicious use of various drugs, but I do wish to emphasize that careful nursing is the all-important item of treatment, and that medicines are often worse than useless, especially if the patient fights against them.

In the general treatment of Stüttgart disease the drugs I most frequently rely upon are carbonate of bismuth and salol, the former as a gastric sedative to assist retention of small quantities of liquid food, and the latter as a general internal antiseptic. These drugs I alternate with brandy in milk. Mouth lesions I dress with iodine solutions and boracic mouth washes. From all accounts my results would appear to be rather more favourable than those obtained by many practitioners, but that may be due largely to the fact that a fair proportion of my patients are crossbreds, and probably possessed of greater stamina.

PAPILLOMATA IN THE DOG.

By G. MAYALL, M.R.C.V.S.

Norwich.

These little tumours may be encountered on the lips, gums and roof of the mouth in dogs. At times they cluster together, and affect a considerable surface on the inside of the upper lip or at the corners of the mouth or lower lip and gums. They may alter the position of a tooth when they are attached to the jaw by a hard firm base.

They are said to often disappear spontaneously, and there is reason for believing this, but when affecting a patient brought for treatment it does not do to accentuate this fact too much. I recently removed about twenty papillomata from the inside of the lips and gums of a Great Dane. Many of the warts of small dimensions were, however, left alone. A spaniel dog, too, with warts all over the roof of his mouth and inside his lips came under treatment; some of these were removed with the scissors. I do not stop here, however, but usually give the dogs iron and arsenic pills internally, and have their mouths swabbed or syringed out with vinegar and water, about equal parts. Both the above-mentioned animals made perfect recoveries, but I had the Great Dane under treatment again shortly after with one of his legs greatly swollen and inflamed from being rubbed with some stimulant liniment. The healthy foreleg measured at the elbow 7 in., and above the carpus 6 in., but the cellulitic one measured 10 in. and 9 in. respectively at these points. I bandaged the whole leg with linen soaked in ammon. chlor. and pot. nit. lotion, and internally gave syrup trifolium co. (Parke Davis) in teaspoonful doses three times daily. After a time the dog made a recovery. It is the first "big leg" I have ever seen in the dog.

PROSTATIC ENLARGEMENT CURED BY CASTRATION.

By FREDK. HOBDAY, F.R.C.V.S.

Kensington, W.

The patient, a valuable old English sheep-dog, aged 10, was for about two years the subject of a very obstinate constipation, which occurred at intervals and caused violent straining. Aperient medicines gave temporary relief, but a large perineal hernia, quite as large as a good-sized cocoanut, eventually developed, and the attacks of pain occurred at more frequent intervals and became more prolonged on each occasion. Rectal examination revealed an enlarged prostate, quite the size of a tangerine orange, and painful on pressure. Treatment by potassium iodide and other drugs not being satisfactory, the owner was advised to allow the operation of castration, and this was performed on January 13. The scrotal wounds healed without any trouble, in fact by primary union, the sutures were removed on the fifth day, and the patient began steadily to improve almost immediately. At the present time (March) the prostate has atrophied, and is less than normal in size, the pelvic hernia is very much reduced and able

to be manipulated without pain, and the attacks of constipation have completely disappeared. The patient has lost the haggard expression he used to have, and is also much improved in bodily flesh. This case is further testimony to the value of the operation of castration for enlarged prostate, and a typical illustration of a number of these cases in old dogs which have come under my notice.

AN INTERESTING OVARO-HYSTERECTOMY.

By FREDK. HOBDAY, F.R.C.V.S.

Kensington, W.

The patient, a very valuable Pekingese bitch, aged 5, had already had two litters of puppies, and was now heavy in whelp again. My attention was first drawn to her in January last, as she was said by the owner to have fainted once or twice and to be apparently losing the use of her hind legs. When I first saw her I diagnosed the symptoms as being due to the very heavy litter of puppies which were distending the abdomen, and, as she was only five days off the date of whelping, I endeavoured by careful nursing, dieting, and the judicious use of stimulants to keep her going along until the auspicious event was actually due. Two days later I was urgently summoned to see her, as the owner thought she would die. She was at times breathing heavily and in a laboured manner, but with care this passed off for another twenty-four hours. On the following day (two days before she was actually due to whelp) she was taken so very ill that I decided to operate immediately, and under the anæsthetic influence of chloroform, ovaro-hysterectomy was performed, eight very fine, well-developed, living puppies being removed. Tincture of iodine was used as the external antiseptic for the skin, and the wound was not touched at all until exactly a week afterwards, when the sutures were removed. The bitch made an uninterrupted recovery, and the puppies were placed with a foster-mother. They lived for five days and appeared to thrive well, but, owing to an unfortunate incident, they were allowed to get in a cold place, and all the litter died in one night.

The case illustrates many interesting points, especially the initial symptoms of collapse before the accouchement was actually due, the number and size of the puppies, and the excellent healing of the wound afterwards without further dressing or interference.

Indexed B. A. 17

Abstracts and Reports.

LEUCOCYTES IN MILK: METHODS OF DETERMINATION AND THE EFFECT OF HEAT UPON THEIR NUMBER.

By H. C. CAMPBELL, V.M.D., M.D.

Pathological Division, Bureau of Animal Industry.[1]

The Composition and Characteristics of Cow's Milk.

Milk contains all the food elements necessary for the development of the young. Not only do these food elements vary as to their percentages in the milk of individuals belonging to any one species, but the variations are still greater between different species. At first thought it may seem easy to give a statement concerning the composition of average cow's milk, but if one studies the subject it will be found that many factors come into play which render it very difficult to determine just what average milk is.

The average chemical composition of cow's milk may be stated to be about as follows: Water, 87 per cent.; fat, 3·9 per cent.; casein, 2·7 per cent.; albumin, 0·7 per cent.; sugar, 5 per cent. Milk also contains cellular elements, the number in normal milk usually not exceeding 500,000 per cubic centimetre.

Just as the chemical constituents have been found to vary according to the period of lactation, health, and breed of the animal, so has the number of cellular elements been found to vary. There is no definite standard of normal milk as to its chemical, physical, and physiological characteristics. In consequence of this the exact line of demarcation between the physiologic and pathologic properties of milk is an arbitrary one and determinable only by careful study.

At the beginning of the first week of lactation cow's milk contains two kinds of cells—colostrum corpuscles and leucocytes. The former are recognized under the microscope by their size and granulations; the latter correspond in morphology to the polymorphonuclear cells of human blood, and are always present in small numbers in normal milk. A third kind of cell, the epithelial cell, frequently gains entrance to the milk in the course of milking, being derived from the teats of the cow or from the hands of the milker.

The Presence of Leucocytes in Milk.

It has long been known that milk contains many bacteria and cellular elements. The relation existing between the kind of bacteria and the cellular elements has been a subject of considerable discussion in the last few years. Early observers found that the leucocytes and bacteria were invariably increased in the milk from a cow with udder inflammation. When milk contained a large number of bacteria and cells, it was thought that some one animal in a herd was suffering from garget or mammitis. Later observers contended that these cellular elements were found in normal milk, and that different animals and even different quarters of the udder in the same animal contained varying amounts of these elements.

[1] *Bulletin* 117.

The variety of bacteria commonly associated with an increase of leucocytes is the streptococcus. Whether the polymorphonuclear leucocyte is to be considered as a pus cell or a normal leucocyte is still an unsettled question. The early milk experts were inclined to consider the presence of large numbers of leucocytes as an indication of udder disease, the reason for this being that they found increased numbers of the streptococci also present. Some later observers—for instance, Savage—did not find this relation. Since normal milk contains leucocytes in varying numbers, it is the writer's opinion that they cannot be considered as pus cells unless they are present in large numbers, associated with pus-producing organisms. The mere presence of leucocytes in milk, therefore, does not seem to indicate pus formation in the udder.

Of the cellular elements found in milk the presence of leucocytes has excited the greatest interest among investigators, especially since Stokes conceived that the centrifugalized sediment containing more than ten leucocytes to a field of a $\frac{1}{12}$ oil-immersion lens indicates the presence of pus in the milk and consequently inflammatory processes of the udder. It is only natural that considerable significance was given to this finding, and since then various investigators have undertaken extensive examinations in order to determine the significance of these leucocytes in milk. The results of the findings of recent observers indicate that the presence of large numbers of leucocytes in milk is indicative of an inflammation of the udder, viz., streptococcic mastitis. Exceptions, of course, will be found where large numbers of leucocytes appear during the first portion of the lactation period and, as some observers contend, when milking is carried on until late in the period of gestation.

The demonstration of a mastitis by the presence of leucocytes would be of very great importance, as it is a well-known fact that such an affection cannot always be established by a clinical examination of the udder, especially in its early stages, and there are even instances when the affection might pass off without disclosing any clinical evidence of its existence. However, with the technique heretofore employed in the numerical determination of leucocytes, there is too narrow a margin between the leucocytes found in the milk of healthy cows and in that of diseased cows to make the majority of these methods of diagnosis satisfactory and practicable.

Various Methods for Determining the number of Leucocytes in Milk.

Different observers have advised various methods for estimating the number of leucocytes in milk in order to establish a standard by which it may be judged. While the several methods recommended are not directly comparable, it is nevertheless evident that an entire lack of harmony exists at present among them, and that the reliability of one or more of the methods is at least doubtful.

The first investigator to adopt a standard was Stokes, of the Maryland State Board of Health. The next was Stewart, of the City Board of Health of Philadelphia. Later Doane and Buckley, of the Maryland Agricultural Experiment Station, devised what is at present considered by many to be the most accurate and uniform method. A similar method was proposed independently by Savage. Many others

have done work along this line, bnt all have used modifications of the methods mentioned.

Three general methods are now in vogue, namely: (1) Smearing the sediment on a glass slide; (2) a volumetric method; and (3) a determination of the percentage of sediment in the milk. The first method in a general way involves the principles suggested by Stokes and Stewart; the second is that originated by Doane and Buckley and by Savage; aud the third method is that devised by Trommsdorff.

All of these methods for determining the number of leucocytes in milk have been used throughout this work, that of Doane and Buckley being slightly modified in some details, as is explained later on. A brief description of the technique used in the methods mentioned follows:—

Stokes's Method.—This consists in placing 10 c.c. of milk in a glass tube, which is whirled in an ordinary centrifuge for ten minutes. The fat and supernatant liquid are then poured off, and one platinum loopful of the sediment is spread over an area of 1 square centimetre on a glass cover slip. This is stained with methylene blue for one minute, and mounted in either water or Canada balsam on a glass slide. Ten fields are counted with a $\frac{1}{12}$ oil-immersion lens, and the average count is taken as the number per field. It has been the custom of those using this method to regard the milk as unfit for use when the number of leucocytes exceeds ten per field.

Stewart's Method.—This method consists in placing 1 c.c. of milk in a small glass tube, stoppered at one end with a rubber plug. The tube is placed on a Stewart disk, with the stoppered end at the edge of the disk, and is centrifuged for ten minutes. The rubber plug is drawn out while the tube is held horizontally, and the sediment on the rubber plug is smeared over an area of 1 square centimeter on a glass cover slip. This is stained for one minute with methylene blue, and is mounted in either water or Canada balsam on a glass slide. Ten fields are counted with the $\frac{1}{12}$ oil-immersion lens and the average count is taken as the number per field. Those using this method are accustomed to regard the milk as unfit for use when the count exceeds twenty-three per field.

Trommsdorff's Method.—This requires the use of a specially constructed tube holding 10 c.c. and drawn out at the bottom into a fine calibre. The narrow part of the tube has twenty graduations, each one representing 0·01 per cent. of the sediment from 10 c.c. of milk. Ten cubic centimetres of milk are placed in the tube, which is whirled in an ordinary centrifuge for ten minutes. At the expiration of this time the tube is taken out and the percentage of sediment read directly from the tube. This method gives the percentage of sediment, but gives no information as to the substances of which it consists. Trommsdorff's method, however, appears to give satisfactory results in practice, and is supported by many investigators as a relatively reliable method for practical use in demonstrating pus in milk.

The Doane-Buckley Method.—This method consists in placing 10 c.c. of milk in a glass tube graduated in cubic centimetres. The tube is centrifuged for ten minutes, and then the fat and supernatant liquid are aspirated off down to the 1 c.c. mark. This remaining cubic centimetre is thoroughly mixed with a saturated alcoholic solution of methylene blue, and is stained for one minute. One drop of this

mixture is placed in a Thoma-Zeiss blood-counting apparatus and the number of leucocytes per cubic centimetre is determined. Those using this method consider the milk below standard when the number of cells exceeds 500,000 per cubic centimetre.

In the experiments here recorded, the writer found it better to aspirate off the fat and supernatant liquid down to the 5 or 6 c.c. mark, being careful not to allow the pipette to go below the surface

TABLE I.—DETERMINATION OF LEUCOCYTES AND BACTERIA IN MARKET MILK BY VARIOUS METHODS.

Number of sample	Number of Leucocytes			Sediment (Trommsdorff)	Number of bacteria	Streptococci	Kinds of bacteria found
	Stokes	Stewart	Doane-Buckley				
	Per field	Per field	Per c.c.	Per cent.	Per c.c.		
1	5·0	10·0	34,000	0·05	9,080	Present	*Staphylococcus albus, Bacillus mesentericus.*
2	4·0	5·0	32,000	·02	877	do.	*Staph. albus, S. aureus.*
3	2·0	2·0	48,000	·01	413	do.	*Staph. albus.*
4	10·0	5·0	64,000	·04	480	do.	*Sarcina alba.*
5	2·0	2·0	32,000	·02	21,000	do.	B. Class IV. (Chester, p. 155).
6	2·0	2·0	32,000	·02	7,500	do.	*Staph. albus, Sarcina pulmonum.*
7	2·0	2·0	120,000	·02	26,000	do.	*B. mesentericus, Mycobact. lactis.*
8	3·0	2·0	64,000	·02	116,000	do.	*Bact. luteum.*
9	3·0	2·0	65,000	·02	76,000	do.	*Staph. aureus.*
10	1·0	2·0	25,000	·02	4,596,000	do.	*Staph. albus.*
11	1·0	2·0	28,000	·01	304,000	do.	*Bact. lactis, B. limbatum.*
12	1·0	2·0	30,000	·03	3,200,000	do.	
13	2·0	2·0	34,000	·02	551,000	do.	*Mycobact. lactis.*
14	1·0	1·0	46,000	·02	64,000	do.	
15	·4	·8	7,000	·02	145,000	do.	*Staph. albus, Micrococcus luteus.*
16	1·0	1·0	34,000	·07	490,000	do.	
17	1·0	3·0	45,000	·03	260,000	do.	*Bact. rugosum, Bact. plicatum, Mic. albus, B. subflavus.*
18	·5	1·0	15,000	·02	30,000	do.	*B. circulans.*
19	2·0	2·0	38,000	·03	200,000	do.	*B. liquefaciens, B. pseudotyphosus.*
20	5·0	7·0	125,000	·05	150,000	do.	*B. mesentericus vulgatus, Mic. acidi lactici, Bact. acidi lacti*

of the liquid. The tube was then filled up to the 10 c.c. mark with distilled water and recentrifuged. This was repeated several times until the upper surface of the liquid was transparent, then the supernatant liquid was aspirated off down to the 1 c.c. mark. The 1 cubic centimetre of sediment remaining was thoroughly mixed, 1 drop of the sediment was placed in a blood-counting apparatus, and the number of leucocytes per cubic centimetre was estimated. The advantage of washing several times and of using no stain was that there were very few fat globules and no precipitate from the stain. With the other method these were frequently found to interfere with the making of an accurate count.

A Comparison of the Methods.—It seemed desirable that a comparison of the foregoing methods should be made. This was done, using the technique above described. In each case there was made a determination of the number of bacteria per cubic centimetre, and the various species of bacteria were identified. Milk from three different sources was examined, namely, (1) market milk; (2) milk from a cow in her first lactation period (the cow had been under observation for some time and was known to have no udder lesions); (3) milk from a

TABLE II.—DETERMINATION OF LEUCOCYTES AND BACTERIA IN MILK FROM COW IN FIRST LACTATION PERIOD.

Number of sample	Number of Leucocytes			Sediment (Trommsdorff)	Number of bacteria	Kinds of bacteria found
	Stokes	Stewart	Doane-Buckley			
	Per field	Per field	Per c.c.	Per cent.	Per c.c.	
1	8·0	3·0	61,000	0·03	1,833	*Micrococcus orbiculatus, Mycobact. pseudo-diphtheria.*
2	1·0	3·0	19,000	·02	8,700	*Mic. orbiculatus, Mic. ovalis.*
3	1·0	1·0	19,000	·02	4,122	*Mic. orbiculatus, Mycobact. pseudo-diphtheria.*
4	·1	1·0	7,000	·01	3,700	*Bacillus liquefaciens, Mic. acidi lactici.*
5	1·0	5·0	8,000	·01	2,833	*Mic. acidi lactici.*
6	·8	4·0	9,000	·01	5,550	*Mycobact. pseudo-diphtheria.*
7	·5	3·0	2,000	·0	1,000	*Mic. luteus, Mic. acidi lactici.*
8	·5	1·0	4,000	·0	1,466	*Mic. ovalis, Mycobact. lactis.*
9	·5	2·0	5,000	·0	2,061	*Mycobact. lactis.*
10	1·0	7·0	3,000	·01	1,505	*Mic. acidi. lactici.*
11	1·5	7·0	10,000	·015	1,600	*Mycobact. lactis.*
12	·3	5·0	8,000	·01	733	*Bact. mycoides, Mic. lactis.*
13	·7	1·0	10,000	·01	811	*Mycobact. lactis.*
14	·6	1·0	12,000	·01	8,360	*Mic. luteus, Sarcina alba, Mic. acidi lactici.*
15	·4	7·0	7,000	·01	927	B. Class II. (Chester, p. 227), *Mic. acidi lactici.*
16	1·0	1·0	14,000	·01	7,000	*B. mesentericus, Sarcina lutea, Mic. acidi lactici*
17	1·0	1·0	16,000	·01	333	*B. mesentericus, Sarcina lactis.*
18	2·0	1·5	14,000	·02	10,000	*B. mesentericus.*
19	4·0	4·0	81,000	·02	672	*B. mesentericus, Mic. aerius, Mycobact. lactis.*
20	4·0	3·0	20,000	·02	16,000	*B. mesentericus, Mic. acidi lactici, Sarcina lactis*

cow with inflammation of the udder. The results are set forth below in tabular form. Table I. gives the results of examinations that were made of market milk.

These samples of market milk were bought from ordinary milk stores in Philadelphia. The various methods used in determining the leucocytes all showed a certain, although not a uniform, relationship; that is, when one ran high the others usually also showed a high count. Streptococci were found either on staining the sediment or on the agar plates in each of the twenty samples examined.

Table II. shows the results obtained from the examination of the milk from a young cow in her first lactation period. The cow had

been under observation for some time and was known to have no udder lesions.

The above samples of milk were taken in a careful manner from a cow at the experiment farm of the Pennsylvania State Live-stock Sanitary Board, in Delaware County. The cow's udder and the milker's hands were washed with a 1 per cent. solution of creolin, and wiped dry with sterile absorbent cotton. The milk was drawn directly from the udder into a sterile glass bottle, which was then plugged with sterile cotton and brought at once to the laboratory for examina-

TABLE III.—DETERMINATION OF LEUCOCYTES AND BACTERIA IN MILK FROM COW HAVING INFLAMMATION OF THE UDDER.

No. of sample	Number of Leucocytes.			Sediment (Trommsdorff)	Number of bacteria	Kinds of bacteria found
	Stokes	Stewart	Doane-Buckley			
	Per field	Per field	Per c.c.	Per cent.	Per c.c.	
1	17	20	425,000	0·03	5,450	*Bacillus mesentericus, B. citreus, Mycobact. pseudo-diphtheria, Mycobact. lactis.*
2	3	5	80,000	·01	300,000	*B. mesentericus, Mycobact, pseudo-diphtheria.*
3	4	12	112,000	·01	9,666	B. Class II. (Chester, p. 227), *Strep. mirabilis, B. rubus.*
4	1	5	14,000	·005	350	*B. aerogenes, Mic. citreus, B. rubus.*
5	4	4	25,000	·005	1,000	*B. mesentericus,* B. Class II. (Chester, p. 227).
6	6	8	95,000	·005	500	*B. aerogenes, Mic. citreus, B. rubus.*
7	10	36	650,000	·03	8,200	*B. mesentericus, B. fluggei, Mycobact. lactis.*
8	50	60	720,000	·04	1.200	*Sarcina lutea, Mycobact. lactis.*
9	11	18	256,000	·01	6,300	*Sarcina lutea, Sarcina alba.*
10	13	19	142,000	·015	250,000	*Mic. acidi lactici, Sarcina lutea, B. columbarum.*
11	7	17	120,000	·015	9,000	Streptococci.
12	6	11	100,000	·01	2,000	*Mycobact. lactis, Sarcina lactis.*
13	13	16	136,000	·015	600	*B. javaniensis,* streptococci.
14	16	17	146,000	·015	9,000	Streptococci.
15	13	24	138,000	·02	800	*B. rubus,* streptococci.
16	3	4	100,000	·01	5,200	*B. rubefaciens, Bact. acidi lacti,* streptococci.
17	6	4	78,000	·01	800	*B. rubus, Sarcina lutea,* streptococci.
	2	4	108,000	·01	166,400	
18	17	17	89,000	·01	3,300	Streptococci.
19						Streptococci.
20	11	11	78,000	·01	2,000	Saccharomyces streptococci.

tion. One sample was taken on every other day, excluding Sundays. In all of the samples the leucocytic count and the percentage of sediment were very low, considered with relation to the standard. No streptococci were found either in an examination of the sediment or on the agar plates.

Table III. shows milk examined in the same manner from a cow having inflammation of the udder.

The above samples were taken from a cow on the same farm and under the same precautions and in the same manner as observed in gathering the milk reported on in Table II.

This cow gave birth to a calf on May 23, 1908. Immediately following this, one quarter of the udder became inflamed and the secretion of milk from this quarter ceased. On June 25, when the first sample was taken for examination, this quarter had become atrophied

TABLE IV.—LEUCOCYTE CONTENT OF HEATED AND UNHEATED MILK FRESHLY DRAWN FROM COW, SHOWING VARIATION BETWEEN FOREMILK, MIDDLE MILK, AND STRIPPINGS.

Number of cow	Parts of same milking	Cellular Content of milk—		Percentage of increase or decrease
		Unheated	Heated to 70° C.	
		Per c. c.	Per c. c.	
800	Foremilk	316,000	2,800,000	+ 786
	Middle milk	440,000	4,000,000	+ 809
	Strippings	572,000	6,200,000	+ 990
800	Foremilk	1,800,000	11,500,000	+ 538
	Middle milk	1,600,000	7,200,000	+ 350
	Strippings	2,800,000	9,600,000	+ 242
708	Foremilk	16,000	45,000	+ 181
	Middle milk	37,000	112,000	+ 202
	Strippings	136,000	236,000	+ 73
800	Foremilk	4,400,000	6,800,000	+ 54
	Middle milk	2,000,000	3,200,000	+ 60
	Strippings	5,200,000	9,600,000	+ 84
708	Foremilk	80,000	1,200,000	+ 50
	Middle milk	380,000	488,000	+ 28
	Strippings	360,000	432,000	+ 20
800	Foremilk	20,480,000	8,400,000	− 59
	Middle milk	1,260,000	2,798,000	+ 122
	Strippings	2,880,000	3,456,000	+ 20
722	Foremilk	128,000	240,000	+ 87
	Middle milk	192,000	392,000	+ 104
	Strippings	660,000	1,180,000	+ 78
806	Foremilk	66,000	25,000	− 62
	Middle milk	30,000	42,000	+ 40
	Strippings	82,000	124,000	+ 51
720	Foremilk	92,000	288,000	+ 213
	Middle milk	80,000	340,000	+ 325
	Strippings	200,000	1,400,000	+ 600
797	Foremilk	160,000	236,000	+ 47
	Middle milk	288,000	597,000	+ 107
	Strippings	488,000	1,158,000	+ 137
797	Foremilk	60,000	156,000	+ 160
	Middle milk	187,000	210,000	+ 12
	Strippings	192,000	344,000	+ 78
761	Foremilk	67,000	240,000	+ 258
	Middle milk	96,000	136,000	+ 41
	Strippings	221,000	312,000	+ 41
716	Foremilk	60,000	176,000	+ 193
	Middle milk	42,000	128,000	+ 204
	Strippings	92,000	320,000	+ 247
718	Foremilk	448,000	1,300,000	+ 190
	Middle milk	310,000	608,000	+ 95
	Strippings	460,000	6,716,000	+ 1,360

and very hard. The samples were a mixture from the other three quarters. The majority of them showed an extremely large number of leucocytes. In 50 per cent. of the examinations streptococci were found either from the sediment or on the agar plates. There seems to

be no doubt that the cow's udder was infected with streptococci. The table seems to show that there is some relation between the presence of streptococci and the number of leucocytes.

The writer has frequently observed that the streptococci which occur with large numbers of leucocytes are in narrow curved chains, and it appears that pathogenic streptococci are usually of this type. On the other hand, some forms of lactic-acid bacteria may be mistaken for small, short, and thick chains of streptococci. Therefore it appears

TABLE V.—LEUCOCYTE CONTENT OF HEATED AND UNHEATED MARKET MILK.

Number of sample	Number of Leucocytes in Milk		Percentage of increase	Number of sample	Number of Leucocytes in Milk		Percentage of increase
	Unheated	Heated to 70° C.			Unheated	Heated to 70° C.	
	Per c.c.	Per c.c.			Per c.c.	Per c.c.	
1	41,000	584,000	1,324	2	42,000	552,000	1,212
	102,000	592,000	480		56,000	506,000	803
	85,000	612,000	620		43,000	490,000	1,039
	64,000	632,000	887		38,000	510,000	1,241
	46,000	626,000	1,263		43,000	540,000	1,156
	70,000	600,000	757	3	50,000	256,000	412
	72,000	636,000	783		62,000	264,000	325
	42,000	608,000	1,347	4	70,000	1,600,000	2,185
	21,000	580,000	2,661		64,000	1,700,000	2,556
	96,000	608,000	533		78,000	1,600,000	2,938
	23,000	616,000	2,578		62,000	1,609,000	2,158
	58,000	604,000	941	5	120,000	312,000	160
	73,000	664,000	809		106,000	308,000	190
	60,000	648,000	980	6	96,000	272,000	183
	34,000	632,000	1,758		108,000	284,000	162
2	102,000	492,000	382		112,000	288,000	162
	74,000	500,000	575		136,000	262,000	92
	100,000	532,000	432	7	60,000	120,000	100
	90,000	512,000	468		32,000	118,000	268
	65,000	498,000	666		46,000	112,000	143
	60,000	468,000	700		58,000	118,000	103
	54,000	516,000	855	8	162,000	222,000	37
	45,000	496,000	1,002		161,000	216,000	34
	40,000	524,000	1,210		156,000	196,000	25
	51,000	504,000	888		164,000	200,000	21
	64,000	496,000	675				

that narrow, curved chains of streptococci occurring coincidently with large numbers of leucocytes should be regarded seriously and as evidence that the milk is unfit for consumption.

DETERMINATION OF LEUCOCYTES IN MILK AFTER HEATING.

During the progress of the work previously described, an article by H. L. Russell and Conrad Hoffman upon "The Effect of Heating upon the Determination of Leucocytes in Milk" appeared in the August, 1908, number of the *American Journal of Public Hygiene*. This paper called attention to the apparent increase in the number of leucocytes in milk following heating. It appeared to be desirable to repeat their work under different conditions. Accordingly the following experiments were made, the details of which are shown in

Tables IV. to IX. The Doane-Buckley method was used by Russell and Hoffman, whereas in our work the modification before described was employed.

In Milk Drawn Directly from Cow.—The first series of comparisons between heated and unheated milk were carried out with milk freshly drawn from the cows. In each instance the foremilk, middle milk, and strippings were studied. The sample was first examined raw; then it was heated to 70° C. and another count of leucocytes was made. The percentage of increase or decrease in the heated sample

TABLE VI.—LEUCOCYTE CONTENT OF DUPLICATE SAMPLES OF UNHEATED AND HEATED MILK.

Number of sample	Unheated			Heated		
	First examination	Second examination	Variation	First examination	Second examination	Variation
	Number	Number	Per cent.*	Number	Number	Per cent.
1	41,000	102,000	59·9	548,000	592,000	7·4
	85,000	64,000	24·7	612,000	632,000	3·1
	46,000	70,000	34·2	626,000	600,000	4·1
	72,000	42,000	41·6	636,000	608,000	4·4
	21,000	96,000	78·1	580,000	608.000	4·6
	23,000	58,000	60·1	616,000	604,000	1·9
	73,000	60,000	17·8	664,000	648,000	2·4
2	74,000	100,000	26·0	500,000	532,000	6·0
	90,000	65,000	27·7	512,000	498,000	3·9
	54,000	45,000	16·6	516,000	496,000	4·0
	40,000	51,000	21·5	524,000	504,000	3·9
	64,000	42,000	34·3	496,000	552,900	11·3
	56,000	43,000	23·2	506,000	490,000	3·2
	38,000	43,000	17·6	510,000	540,000	5·8
3	50,000	62,000	19·3	256,000	264,000	3·0
4	70,000	64,000	8·5	1,600,000	1,700,000	5·8
	78,000	62,000	20·5	1,600,000	1,400,000	12·5
5	120,000	106,000	11·6	312,000	308,000	1·2
6	96,000	108,000	11·1	272,000	284,000	4·1
	112,000	136,000	17·6	288,000	262,000	9·0
7	60,000	32,000	46.6	120,000	118,000	1·6
	46,000	58,000	20·6	112,000	118,000	5·0
8	162,000	161,000	·6	222,000	216,000	2·2
	156,000	164,000	4·8	196,000	200,000	2·0

* The smaller number is used as the basis in each case.

is shown in the last column of Table IV., which gives the results in detail.

In the forty-two samples here reported, forty show that a much larger number of cells was found in the heated samples than in the unheated, while only two showed a decrease. The figures seem to indicate that heating causes a large increase in the number of leucocytes found on examination. The reason for this condition Dr. Russell explains in the following manner: "When milk is heated for ten minutes at 60° C. the creaming power is greatly diminished. The fat-globule clusters are broken down, and the butterfat is more or less homogeneously distributed throughout the milk serum. These fat-

globule aggregations rise steadily to the surface and probably entangle a good many of the cell elements; but with the more homogeneous emulsion of the fat due to the action of the heat the leucocytes are probably not enmeshed, and are therefore free to respond to the action of gravity."

The writer concurs in this explanation, since from the very nature of the leucocyte cell there can be no multiplication of these cells and therefore no actual increase in the number of leucocytes. A larger number is found in heated than in unheated milk, because they more freely settle with the sediment as a result of becoming disentangled from the fat globules, due to the effect of the heat. In unheated milk, on the other hand, only a comparatively small proportion of the leucocytes settle with the sediment.

In Single Samples of Market Milk.—The next table (Table V.) shows a number of counts made from single samples of market milk. In each case a number of subsamples were examined, but only one

TABLE VII.—LEUCOCYTE CONTENT OF MILK AT DIFFERENT TEMPERATURES.

Number of samples	15° C.	30° C.	40° C.	50° C.	60° C.	70° C.	80° C.	90° C.	100° C. for five minutes
	Per c.c.	Per c.c.	Per c.c.	Per c.c.	Per c.c.	Per c.c.	Per c.c.	Per c.c.	Per c.c.
1	70,000	80,000	140,000	158,000	1,200,000	1,600,000	1,600,000	—	—
	64,000	92,000	148,000	120,000	1,096,000	1,700,000	1,440,000	—	—
	78,000	84,000	144,000	154,000	1,120,000	1,600,000	1,640,000	—	—
	62,000	58,000	132,000	150,000	1,056,000	1,400,000	1,560,000	—	—
2	60,000	40,000	76,000	128,000	184,000	196,000	198,000	576,000	920,000
	48,000	32,000	100,000	132,000	176,000	194,000	200,000	592,000	900,000
	36,000	38,000	104,000	140,000	204,000	204,000	192,000	600,000	980,000
	40,000	46,000	108,000	164,000	188,000	208,000	196,000	630,000	1,000,000
3	144,000	136,000	132,000	140,000	184,000	256,000	704,000	724,000	900,000
	124,000	112,000	104,000	146,000	180,000	258,000	640,000	716,000	1,086,000
	106,000	124,000	128,000	144,000	186,000	260,000	716,000	680,000	946,000
	104,000	116,000	114,000	146,000	188,000	244,000	648,000	728,000	1,216,000
4	36,000	26,000	42,000	126,000	140,000	170,000	356,000	660,000	600,000
	38,000	36,000	56,000	130,000	148,000	154,000	368,000	580,000	610,000
	18,000	44,000	78,000	120,000	139,000	158,000	360,000	670,000	630,000
	42,000	28,000	60,000	124,000	145,000	144,000	380,000	590,000	650,000

count was made from a given tube. For each count throughout all the work shown in this table a new tube was taken, centrifuged, and examined.

In this, as in Table IV., the heated sample in every case shows a marked increase in the number of leucocytes, and in many cases the increases are large.

In Duplicate Samples of Milk.—In the next table the results of the examination of a number of duplicate samples are shown. A study of this table shows that the analytical error is much smaller in samples heated to 70° C. than in unheated samples.

Of the twenty-four duplicate samples examined, unheated and heated, the average variation in the unheated was 27·1 per cent., as compared with only 4·2 per cent. in the heated samples. These figures would seem to indicate that in routine inspection of milk one should heat the milk to somewhere near 70° C. before making an examination for leucocytes.

Number in Milk Heated to Varying Temperatures.—After noting the above results a number of samples were examined at different temperatures, in order to observe at what point the change in the apparent leucocytic content occurred. The results are set forth in Table VII.

The most satisfactory results were obtained between 60° and 70° C. There was an increase at 80°, 90°, and 100° C., but the results were not so uniform and the examination was much more difficult to make on account of the coagulated albumen, which interfered with the count. At the high temperatures the leucocytes were more or less disintegrated and could not be so easily differentiated from foreign material, hence the count was rendered less reliable.

Number in Milk After Pasteurizing and Cooling.—In pasteurized milk the leucocyte count is usually higher than in raw milk; therefore it

TABLE VIII.—LEUCOCYTE CONTENT OF MILK BEFORE AND AFTER HEATING AND REHEATING.

Number of sample	First examination		Second examination (after cooling at 15° C. for twenty-four hours)	
	Unheated	Heated to 70° C.	Heated and cooled	Heated second time to 70° C.
	Per c.c.	Per c.c.	Per c.c.	Per c.c.
1	30,000	430,000	416,000	404,000
	19,000	428,000	424,000	412,000
	28,000	436,000	428,000	416,000
	26,000	432,000	360,000	422,000
2	260,000	380,000	452,000	348,000
	226,000	384,000	420,000	340,000
	208,000	380,000	440,000	346,000
	288,000	382,000	460,000	320,000
3	80,000	212,000	236,000	196,000
	60,000	204,000	228,000	204,000
	92,000	224,000	240,000	216,000
	76,000	222,000	208,000	200,000
4	60,000	248,000	214,000	268,000
	74,000	250,000	204,000	240,000
	78,000	240,000	216,000	264,000
	96,000	256,000	208,000	270,000

was thought desirable to examine a series of four tubes from the same sample. The first tube was unheated; the second was heated to 70° C.; the third was heated and then cooled, and kept at 15° C. for twenty-four hours; the fourth was heated, then cooled, kept at 15° C. for twenty-four hours, and then heated again. The purpose of these experiments was to see if the milk after being pasteurized and cooled returned to the same condition as regards the number of leucocytes as that in which it was found before pasteurization.

Table VIII. gives the results of these experiments, and indicates that there may be here a principle upon which may be based another method for determining whether milk has been heated or pasteurized.

The table shows that in every instance after the milk was once heated the cooling did not bring it back to its original condition,

while the second heating to 70° C. did not increase the count over the first heating.

Determinations by Various Methods.—The writer's modification of the Doane-Buckley method, which was considered the most accurate, was the only one used in the routine determinations of the number of leucocytes found in heated milk, as reported in Tables IV. to VIII., inclusive. For the sake of comparison, therefore, a number of samples were examined in similar manner by the other methods—namely, those of Stokes, Stewart, and Trommsdorff. The results which are shown in Table IX. indicate that there are also more leucocytes found in heated milk than in unheated milk by each of these methods of determination.

TABLE IX.—LEUCOCYTE CONTENT OF HEATED AND UNHEATED MILK, AS DETERMINED BY STOKES, STEWART, AND TROMMSDORFF METHODS.

Number of sample	Milk Unheated			Milk Heated to 70° C.		
	Stokes	Stewart	Trommsdorff	Stokes	Stewart	Trommsdorff
	Number per field	Number per field	Per cent. sediment	Number per field	Number per field	Per cent. sediment
1	0·6	1·8	0·005	5·1	13	0·03
	·5	1·9	·005	10·1	12	·03
	·2	2·7	·005	8·0	7	·03
	·3	1·0	·005	4·0	10	·03
2	3·3	13·0	·02	6·0	24	·03
	3·5	14·0	·02	15·0	22	·03
	3·7	9·0	·02	8·0	15	·03
	1·8	10·0	·02	12·0	12	·03
3	·3	1·5	·01	3·0	6	·02
	·2	·5	·01	2·7	4	·02
	·1	1·6	·01	4·3	8	·02
	1·0	1·0	·01	6·0	5	·02
4	·2	·1	·01	·4	3	·02
	·1	·15	·01	·5	2	·02
	·1	·2	·01	·3	6	·02
	·15	·1	·01	·6	4	·02

CONCLUSIONS.

(1) The effect of heating upon the number of leucocytes found in milk increased the number of leucocytes per field in Doane and Buckley's, Stokes's, and Stewart's methods, also the percentage of sediment in the Trommsdorff method, in all of the samples examined.

(2) It appears that heating is as essential as any other part of the technique in determining as nearly as possible the number of cellular elements in milk.

(3) Since heating greatly increases the number in the count of the leucocytes, it seems necessary that a higher leucocyte standard shall be adopted in judging milk.

(4) Milk inspection may be greatly benefited by the establishment of some rational standard for the leucocyte content of milk, but more study must be given to this subject in order to obtain the desired knowledge.

THE NEED FOR ORGANIZATION AND EDUCATION IN THE MEAT TRADE.

By LOUDON M. DOUGLAS.

Lecturer on the Meat Industry, Edinburgh.

THERE was a time, indeed, when the meat industry was better organized than it is even at the present day. That was the time when the trade guilds were in existence, but in the year 1835 these trade guilds were abolished, and with them the only organization in connection with the meat trade. These trade guilds had high privileges, and the members of the meat industry, who had their own guilds along with the other industries which existed in the various towns of the country, were really responsible for the election of the Town Councils. Our modern system, however, is totally different, and perhaps it is as well that it is so, as in these old times there is no doubt but that the guild privileges were abused. At the same time I may say that not so very long ago the members of the meat industry had no status at all.. I think I am correct when I say that there is a statute existing in England, which enacts that no meat purveyor shall be allowed to sit on a jury where life is concerned, and they suffered other disabilities because of their calling. The abolition of the organization, therefore, in the year 1835, was a step backwards, and it was not rectified until comparatively recent years.

It was in the year 1888 that the Meat Traders' Federation came into existence, and since then it has done much to advance and protect the interest of all meat traders. At the present moment the number of members is something like 20,000, and a strong force in the country.

Few people realize what an enormous amount of money is invested in the meat industry, and how many countries rely upon British markets for outlets for their surplus meat products. Last year, for example, we imported nearly 600,000 tons of beef, mutton, and lamb, and it is estimated that our home supply amounted to 1,128,000 tons during the same period—figures which represent a gigantic amount of human industry, and also involve many millions of pounds in capital. It is, therefore, surely not an unreasonable proposition that a trade which has such great interests should be represented in the Parliament of the country.

Warranty.—One of the questions which the National Federation has had to consider is that of warranty. It has always seemed to me preposterous that the meat purveyor who goes into the market with his cash in his hand, and buys what is apparently a sound bullock, should have to lose the value of that bullock, or any part of it, if it should turn out to be diseased. It seems to me that the use of the tuberculin test would establish the fact whether such a bullock was sound or not, and the onus of ascertaining that fact should lie upon the farmer, as there is no ready means whatever of ascertaining whether a bullock is diseased or not when it is exposed in open market.

I do not mean to say that there is nothing to be said for the farmers' position, as it is quite within the knowledge of everyone here, I dare say, that many farmers are not breeders of their own stock,

but are simply feeders, and consequently they run the risk of purchasing stock which are diseased when handed over to them. At the same time, I do not see why such stock should not be subjected to the tuberculin test. Naturally, however, it will be asked who has to pay for such animals as are discovered to be diseased by the tuberculin test? And the answer to this is that such funds as are needed must be provided by the State.

Veterinary Department.—This leads me to say that I think the time has arrived for the establishment of a meat industry department in connection with the Board of Agriculture. There is very little understood about the meat industry by the Board of Agriculture at the present time. I mean by that, that it is not a specialized department as are others, and consequently suffers from want of attention. As I have already mentioned, the meat industry has vast interests, not only in this country, but in connection with the imports of foreign supplies, and there ought to be a Department of State instituted which wonld have the full control not only from the veterinary, but from the industrial point of view of the meat industry.

Public Abattoirs.—There is another section of the meat industry which, I think, may be administered in the same way, I mean public abattoirs. Such institutions are bound to become universal in the future, but it is a pity if the private slaughterhouse owner should be penalized and lose the power of his vested property and rights without compensation. Private slaughterhouses are in many cases constructed so as to meet every requirement of hygiene, but as a general principle it may be laid down that the control of the meat supply can be better carried out in public institutions, and I believe that the majority of the members of the meat trade are of opinion that public abattoirs would certainly lend themselves to easier veterinary inspection. The question therefore resolves itself entirely into compensation for the private slaughterhouses in existence. When you read the returns of the imported meats into this country from the British Colonies, Argentina, the United States, and many other countries, you will be apt to enquire if it is worth while putting down public abattoirs, considering that the home trade is threatened with extinction by reason of foreign competition. That, however, is a very wide question, which leads me to say that if foreign nations are able to send us meat supplies cheaper than we can produce these at home, it is because they have made it their business to acquire a thorough knowledge of the technique of the business, and they are thereby able to utilize the by-products to the very best advantage. Some of these nations have specialized meat products to such an extent that they obtain something like 100 different commodities from a bullock. As a consequence the principal articles of beef and mutton are bound to be rendered cheaper by the fact of the increased profits from these by-products. In this country there is no such development of the meat industry, and such by-products as I refer to are either inadequately dealt with or are wasted altogether.

This brings me to a matter in which I am personally much interested—namely, the question of technical education, and I do not see why there should not be organized courses of instruction for members of the meat trade. I can personally testify to the fact that where such courses of instruction have been offered, such as in the City of Edinburgh, they have been taken advantage of with the greatest

possible enthusiasm, and I am certain that if the National Federation were to devote itself to the organization of courses of instruction throughout the country, they would confer a lasting benefit on the trade. The meat trader requires to be educated up to his business—I mean the technique of his business—as nowadays he must be not only a meat purveyor, but a little bit of a veterinary surgeon; he must also be something of a chemist and a bacteriologist, if he would understand thoroughly his business, and such understanding would put him in such a position that he would be able to utilize his by-products to the best advantage. The outlook, indeed, is such that closer organization and higher education are both needed in connection with the meat industry, and both of these things will bring great rewards, not only to the individuals themselves, but to the country at large.

Reviews.

PATHOLOGIE CHIRURGICALE DE L'APPAREIL DIGESTIF (Surgical Pathology of the Digestive Apparatus). By C. Cadéac, Professor at the Lyons Veterinary School. 1 vol., 520 pp. 186 figures in the text.

This handy little book is published by J. B. Baillière et fils, 19, Rue Hautefeuille, Paris. It is one of a series which Professor Cadéac has produced in his "Encyclopédie Vétérinaire," the others including the surgical pathology of the foot, skin, blood-vessels and lymphatics, tendons, muscles, nerves, and articulations.

The ailments of the mouth, pharynx, œsophagus, stomach, and intestine are dealt with, and in many cases illustrated. Horses, bovines, sheep, and pigs each have their general and special defects and ailments in the mouth and throat region. The study of dental complaints comprises not less than 100 pages. The terrible ravages of epithelioma affecting the roof of a horse's mouth is an interesting illustration, so also are those representing tympanites of the guttural pouches and harelip in the dog, before and after operation (Hobday).

There is no work which veterinarians have resented so much being deprived of as a treatise on the surgical pathology of the domesticated animals.

M. Cadéac has been collecting material for the last ten years, and has made a reasoned synthesis of it. He studies each organ in a chapter comprising in its turn a series of articles showing the types of alteration that this organ may undergo.

The order of classification of all complaints is the anatomical order.

The domestic animals differ from an anatomical point of view, and there are correlative differences in their pathology. Each species of animal has its illnesses. It was urgent to have a pathology for each animal. M. Cadéac's classification admits of dividing, characterizing, and differentiating the pathologies, and shows the relationship that exists among them.

The work is illustrated with numerous figures, which add to the

clearness of the descriptive text. The book is tastefully bound with a little bit of that French *chic* and *distingué* appearance that adds to the appearance of volumes.—G.M.

PATHOLOGIE CHIRURGICALE DES ARTICUATIONS. By C. Cadéac, Professor de clinique a l'école vétérinaire de Lyon. Published by Baillière et Fils, 19 rue Hautefeuille, Paris. Price 12 fr.

As far as we know this is the only veterinary work which deals entirely with the numerous affections to which the articulations of the domesticated animals are liable.

Text-books on general surgery cannot adequately deal with what may be regarded as special branches, without being enlarged to an inordinate size, and need for specialization is one that is evident to all.

Professor Cadéac's present contribution will at least help to fill in one serious blank. This work contains 442 pages of matter with 142 illustrations. We trust that the time is not far distant when English translations of it will be obtainable.

The various forms of arthritis are concisely treated, and, as is true of many other French works, in a manner easily understood by anyone possessing even a slight acquaintance with that language.

On page 257, Cadéac, in referring to arthritis of the hock, says "the hock is the predilection seat of all forms of arthritis. Those cases not caused by traumatic influences are localized by work and fatigue." Following this statement we find forty-eight pages dealing with hock affections. The author recognizes all the common agencies regarded as predisposing factors to hock disease, and also describes the ones which are generally held to be exciting causes. Among the latter, however, some may be inclined to think that rather too much stress has been laid on the part played by micro-organisms in joint disease. In the above-mentioned article the following will be found: "he who says arthritis, says infection of a joint or the intervention of toxins which alight there."

Most of us have not been in the habit of regarding all cases of osteo-arthritis of the hock as being due to the activity of micro-organisms, and, although we know that many hock affections are caused by bacteria, or are complicated by them, we feel that perhaps Professor Cadéac is a little too enthusiastic in the support of his suggestion.

However, no one who reads the book will be disappointed. It is concise, full of interesting matter, and inexpensive.—M.J.

Miscellaneous.

UNIVERSITY OF LONDON.

FACULTY OF SCIENCE (VETERINARY SCIENCE). GENERAL INTERMEDIATE EXAMINATION (PART I.) FOR INTERNAL STUDENTS.

PASS LIST, JANUARY, 1910.

101. Horton, Frank Fielding, Royal Veterinary College.

Examiners: Organic Chemistry: A. W. Crossley, W. P. Wynne, A. McKenzie, J. Wilson. Veterinary Anatomy: O. C. Bradley, E. S. Shave. Biology: J. G. Kerr, W. H. Lang, F. W. Oliver, W. G. Ridewood, F. Buchanan, F. E. Fritsch, D. T. Gwynne Vaughan, H. W. Unthank.

THE VICTORIA UNIVERSITY OF MANCHESTER.

EXAMINATION RESULTS, MARCH, 1910.

THE following gentlemen have obtained the Diploma in Veterinary State Medicine:—

Henderson, W. W., M.R.C.V.S.	Mattinson, A. B., M.R.C.V.S.
Holroyd, John, M.R.C.V.S.	De Vine, Brennan, F.R.C.V.S.

Translations.

CRIB-BITING IN RUMINANTS.

BY STAFF VETERINARY-SURGEON KULL.

IN general crib-biting is only noticed in the horse, ass, and mule tribe, and is considered more or less injurious to them. In the law-book of this country it is entered as an unsoundness. Crib-biting in ruminants has seldom been noticed, and on that account the following case may be of interest:—

During the last manœuvres I watered my horse during a critical pause at a small isolated homestead in the Schwetz district. The well was situated near the entrance to a cow-house, and during a pause in the pumping I heard a distinct crib-biting noise in the standing. I went in expecting to see a crib-biting horse, but was much astonished to find only three cows. One of them—roan and rather thin—cribbed by drawing down the bottom jaw and at the same time putting out her tongue on the anterior edge of the manger. The sound which the swallowing down of air caused resembled exactly that produced by a bad crib-biter. The owner told me the cow was seven years old; that he himself had bred her, and that the defect had considerably increased during the last two years. As the cow had given little milk for some months, and kept getting thinner, the owner wanted to sell her.

This case shows that crib-biting not only may occur in cattle, but that it may become a very pronounced unsoundness.

(*Zeitschrift für Veterinärkunde.*)

SCOPOLAMINE AND ITS ACTION AS AN ADJUVANT IN CHLOROFORM ANÆSTHESIA.

By GEORGE M. ILIESCO.

Principal of the Veterinary College at Bucharest.

Scopolamine is an alkaloid found in the root of the plant *Scopolia japonica*, in the seeds of *Hyoscyamus niger* and of *Datura stramonium*, in the leaves of *Duboisia*, and in small quantity in the root of *Atropa belladonna*.

According to Lademburg, scopolamine, hyoscine and atropine are three identical substances, having all the formula $C_{17}H_{23}NO_3$.

Scopolamine was isolated by Schmidt in 1892, and later was studied by Pawloff, Claussen, and the pupils of Kobert, who found that this alkaloid exercises a sedative action on man.

According to Guauck, Claussen, Sorkt, Wood, and Ernst, scopolamine produces somnolence in man ten to twelve minutes after its administration.

Ernst showed that large doses produced excitement in the dog, and that this alkaloid is relatively little toxic for warm-blooded animals.

Paukul advises injections of bromhydrate of scopolamine to revive animals in chloroformic syncope.

Schneiderlin recommends scopolamine, associated with morphine, in the form of subcutaneous injections to produce sleep.

In 1897 De Stella made a detailed experimental study with this substance. He found that cold-blooded animals (such as frogs) resisted considerable doses, the fatal dose being $\frac{1}{200}$ of its weight. The dog is killed by 0·66 grm. per kilo of its live weight, whilst the rabbit resists much stronger doses: 1 grm. per kilogramme of the live weight. In frogs small doses do not accelerate the heart-beats; toxic doses produce paralysis of the heart. In the dog small doses cause acceleration of the heart and increase blood-pressure-phenomena accentuated by increasing the quantity given; whilst big doses produce contrary effects.

Scopolamine diminishes salivary secretion in the dog, biliary secretion in the rabbit, and diminishes the quantity of urine in the two species; its employment is therefore dangerous in renal affections.

In 1899 Bourgon made a communication to the Biological Society relative to a case of idiosyncrasy to scopolamine. He recommends this alkaloid, however, as a very important mydriatic, especially where there is a tendency to glaucoma.

Another important study on scopolamine has been made by Kochman at the laboratory of Jena, and his results agree with those of De Stella. Towards the end of 1905 Kouffart and Walravens published a work on "The Employment of Scopolamine as a General Anæsthetic in Surgery," in which they affirm that the principal quality of this special narcosis is to much reduce the quantity of chloroform administered.

Laurendeau, from the good results he had obtained, advised the employment of scopolamine in obstetrics.

Challet observed hypnotic and sedative effects in mental maladies.

In 1906 Professor Dupuis, helped by his assistant Van den

Eeckhout, observing certain successes obtained in human medicine by employment of this alkaloid, carried his observations further to the large domestic animals.

They discovered in their researches that scopolamine constitutes a valuable help in anæsthesia by chloroform, and that this mixed method, "scopolamine, *plus* chloroform" has advantages which no no other method possesses.

Comparing anæsthesia by chloroform alone, with that by chloroform *plus* scopolamine, we found:—

(1) The period of initial excitement produced by chloroform when administered alone, which may be long, violent, and often dangerous, is almost suppressed by the employment of scopolamine.

(2) The quantity of chloroform employed in the mixed method of chloroform *plus* scopolamine is smaller than in the case of anæsthesia by simple chloroform.

(3) The time occupied is shorter with mixed anæsthesia than chloroform alone, and the recovery of the animal more rapid.

Thirty experiments were made, and the scopolamine was administered intravenously, but it may be equally as well used subcutaneously. For these injections the solutions may be made in distilled water. For intravenous injections Locke's liquid (without glucose) has been used as the vehicle. The solutions may be made in the proportion of 1 in 200, 1 in 100, and even 1 in 50.

The dose employed varies according to the weight and temperament of the animal. The effects may be obtained by weak doses, $\frac{1}{10}$ milligramme suffices for the frog, and for the dog and horse 2 to 6 milligrammes per kilogramme of the body weight. All causes which diminish the quantity of chloroform necessary to produce anæsthesia lessen danger of cardiac syncope. Cardiac syncope has been attributed to excitation of the pneumogastric nerve or its bulbar nucleus by chloroform. It is said that by paralysing the pneumogastric scopolamine prevents syncope, but this we doubt.

Conclusions.

(1) Scopolamine accelerates the heart-beats, augments blood-pressure, increases respiratory movements. It modifies the peripheral nervous system, suppressing the pneumogastric in the dog and moderating its action in the rabbit. One does not observe any appreciable modification of the sympathetic; it has no action on the depressor nerve. It increases the excitability of the nervous centres at first, and depresses them afterwards.

(2) Scopolamine is an important auxiliary in chloroform anæsthesia, this effect being obtained in a shorter time and with less chloroform.

(3) This action of scopolamine is due to two modifications on the system: (*a*) On the central nervous system; (*b*) on the circulation and respiration.

(4) Fatigue affects anæsthesia by chloroform in the same way and by the same mechanism as scopolamine.

(5) The quantity of chloroform employed being much reduced, the cerebral troubles produced disappear rapidly, the animal wakes quickly, and sensibility is rapidly re-established. Only dilatation of the pupil remains for twenty-four hours, and sometimes longer.

(6) Scopolamine prevents cardiac syncope, not by paralysing the

pneumogastric but by reducing the quantity of chloroform employed; one thus avoids heart intoxication.

(7) Mixed anæsthesia by chloroform *plus* scopolamine is recommended on one condition only; one must not push anæsthesia too far, for respiratory syncope once produced, the heart being very accelerated under scopolamine, the oxygen of the blood is rapidly consumed, and the animal dies more quickly than in the case of anæsthesia without scopolamine.

(*Arhiva Veterinaria.*)

LACERATION OF THE MASTOIDO-HUMERALIS AND OF THE ŒSOPHAGUS IN A HORSE.

By B. GERMANI.

The horse, Mercury, when seen one evening, showed the following symptoms: Refusing food and drink; in the morning there was slight tumefaction between the middle and lower third of the inferior region of the neck. Tumefaction increased rapidly, and at the time of the visit the swelling extended from the larynx to the entrance of the chest, and rose up laterally to the jugular furrows. Besides, the animal presented some general symptoms: head extended, coat upstanding, muscular tremors shown, gait uncertain. Temperature 39·4° C.; mucosæ injected. The tumefied region was hot and tender; the skin bore no trace of contusions; there was certainly a traumatic lesion, but exploration of the deep organs was impossible on account of the swelling..

Scarifications, followed by the application of warm antiseptic compresses, were practised in the tumefied region, and gave exit to an abundance of citron-coloured liquid. The œdema, which had diminished at first, rapidly increased in the evening, and asphyxia threatening, tracheotomy was performed. The animal died on the evening of the following day. At the autopsy, besides œdematous infiltration of the whole tumefied region, there was an interesting rent in the mastoido-humeralis muscle, involving two-thirds of its extent, situated between the middle and inferior third of the neck. At this level there was a large pocket the size of a nut containing a blackish, hæmorrhagic mass in which a mixture of putrefied alimentary *débris* was found. The œsophagus had an elliptical rent with fringed borders, the muscular tunic friable and gangrenous, the mucosa not notably altered. The connective tissue around the œsophagus was infiltrated with blood and contained alimentary particles.

The cause of the lesions was supposed to have been the bite of a neighbouring horse, which had been inflicted when the muscle was contracted, and whilst the œsophagus was traversed by an alimentary bolus.

(*La Clinica Veterinaria.*)

APPLICATION OF BIER'S METHOD IN TWO CASES OF TRAUMATIC ARTHRITIS.

By MEYRAUX and LANCELEUR.

The authors tried passive hyperæmia by venous stasis in two extremely grave cases of arthritis, which ended in cure in one case and slaughter in the other. The first case was interesting. Instead of slaughtering the wounded animal at once, which had an open hock and fetlock joint, due to wounds in embarkation, the authors tried Bier's method. They applied a linen bandage 0·05 metre in width and 3 metres long above the hock, moderating compression, but enough for the finger to pass easily between the skin and the bandage. This was left in place from 7 to 11 o'clock in the morning, and from 3 to 9 o'clock in the evening for eight days. The wounds were not treated.

On the morning of the third day they were surprised to find pain much less.

The synovial discharge rapidly diminished and was dried up seven days after entrance of the sick horse into the infirmary. There remained only two large benign, granulating wounds, which were treated ordinarily.

(*Revue Générale de Médecine Vétérinaire.*)

AN INTERESTING CASE OF ANTHRAX IN THE COW.

By Dr. OPPERMANN.

Wanzleben.

In May I was called to a cow suddenly taken ill. As I could not get to the animal until two hours afterwards, I found her necessarily slaughtered. The owner told me that the cow in question was 6 years old, well nourished, had eaten well the evening before, and given her accustomed quantity of milk; on the following morning she had refused food, and only given a litre of milk; patient was then slightly blown up and weak. The temperature taken by the owner at the time was 38·5° C. The advancing weakness of the cow and the loud moaning led to the belief that the illness was due to a foreign body, and she was therefore slaughtered. Hæmorrhages from the natural openings of the body or swellings were not observed.

The *post mortem* showed flesh well bled. Blood dark red, clotting badly. No changes on the subcutaneous tissue. Under the endocardium of the left ventricle numerous hæmorrhages as large as a penny piece. Lungs well retracted, dark rosy red. Liver hard as a board from distoma invasion. Edges of the spleen slightly rounded. Spleen scarcely enlarged, brownish red, pulp browny red, not liquescent. Kidneys dark brownish red. Intestinal glands swollen and greyish white. Under the duonenal mucous membrane numerous hæmorrhages of small size and punctiform. Lymphatic glands slightly swollen and sodden, and partly studded with punctiform hæmorrhages. The submucosa of the abomasum was disintegrated, gelatinous, and swollen in some places to 4 cm.; the jelly-like parts showed streaky hæmorrhages.

I took a piece of spleen, kidney, and mucous membrane of the abomasum home, and by bacterioscopic coloration with safranin found numerous typical anthrax bacilli in all three specimens. Macroscopically the case might have been taken for one of septic inflammation of the stomach, which is not uncommon in cattle.

This case shows, however, that a certain diagnosis is only possible by microscopic examination; had such not been undertaken, the flesh as well as the skin would have been used, and disinfection of stall, slaughter place, and instruments neglected.

(*Deutsche Tierärztliche Wochenschrift.*)

A CASE OF OSTEOMALACIA IN THE HORSE.

By Chief Veterinary-Surgeon Dr. SUSTMANN.

Dresden.

A service horse, aged 8, which had previously been a bad doer, fell under notice for about a month on account of languor and listless gait: His condition was poor. In spite of careful and repeated examination, a result of positive value could not be established. Opportunity for grazing was present, and the horse was turned out several hours daily. He was seldom worked, and then only lightly.

After such an event he one day suddenly, without any warning, showed bleeding from the nose and tears from the eyes. A general examination was again negative. On percussion of the cavities of the head, the upper maxillary sinus on the right side gave a hollow sound. At the same time, the thin, bony plate began to bulge. Trepanation of the cavity revealed nothing special; but the tears ceased and the wound had almost healed in four weeks.

Ophthalmo reaction for the diagnosis of tuberculosis, was without reaction. Microscopic examination of the blood (staining with Ehrlich's triacid solution), as far as its corpuscular elements were concerned, only showed this deviation from the normal, namely, that the leucocytes showed exclusively the polynuclear form.

About six weeks after trepanation, bulging of the frontal bone occurred, and over the frontal sinus on the right side there was a swelling as big as a walnut. Osteomalacia was now diagnosed, and corresponding treatment adopted. Suitable and varied food of every kind was now given, and a mixture of phosphate of lime (a teaspoonful in in each feed), together with rest and exercise, prescribed.

About nine months from the commencement of the illness the horse was ridden out for trial for half an hour. When brought back and put in the box he could not stand, even when supported. As no improvement occurred he was killed.

The *post mortem* showed typical osteomalacia. The upper jaw, breastbone, and ribs could easily be cut through with a knife. The external layer of the bone was covered at certain places with red punctæ, and the bone-marrow was red and partly fluid. The cartilages of the joints showed more or less violet or bluish tinge. The other bones were still hard, but showed single punctiform hæmorrhages on the sawn surface.

(*Zeitschrift für Veterinärkunde.*)

INTRODUCTION OF GLANDERS THROUGH ENGLISH HORSES.

By Dr. STÖDTER.

Of twenty-nine English horses imported into Denmark some time ago, seven were found to be affected with glanders soon after arrival. This cannot be wondered at when we hear what the first veterinary authority in England, Sir John McFadyean, reports in the *Journal of Comparative Pathology and Therapeutics* (vol. xviii., p. 23): In the year 1892 there were 3,001 cases in Great Britain; in 1898, or six years later, 1,472, and in 1904 no fewer than 2,658 cases. Glanders has spread all over Great Britain in consequence of insufficient veterinary police regulations. Most cases are to be found in London, Liverpool, Manchester, Birmingham, and Glasgow. It is important to note that the greater number of these cases were clinically recognizable. McFadyean accepts that about 1,000 other horses in Great Britain are affected with occult glanders—truly a sorrowful picture! One must strongly agree with Bang when he says that the purchase of English horses, especially such as are used in London, is an extremely dangerous thing, when considered from the point of view of veterinary police. Let us hope that the English Glanders and Farcy Order, soon to come into force, will effect a change.

(*Maanidschrift for Dyrlaeger.*)

PIROPLASMOSIS IN DOGS.

By Chief Veterinary-Surgeon STAHN.

A fox-terrier bitch, aged 4, belonging to a breeder, fell ill with catarrh of the stomach and intestines, but was not feverish. On the following day her condition had improved; but a three-months-old dog of the same breed showed the following symptoms of illness: Pronounced yellow colour of the visible mucous membranes and skin, especially on the belly and internal surfaces of the thighs; lassitude and weakness of the hind-quarters, decline of appetite. The greatly strengthened heart-beat was not evident; but, on the other hand, the pulse was weak. A feverish rise of temperature was absent. The urine was coloured reddish-yellow to yellowish-brown.

After the introduction of the illness a few weeks elapsed, and then two dogs of a like age died in a few days exhibiting the same symptoms of illness. Veterinary treatment of the patients was not undertaken, nor was a *post mortem* made, the owner considering the illness a simple gastro-intestinal disease.

In view of a short notice in the departmental press on the occurrence of piroplasmosis in dogs, I took some sterile blood from an aural vein with the object of examining it for blood parasites. Staining by Giemsa's method gave negative results (a short time previously examination for *Piroplasma bigeminum* was also without result). On the contrary, staining with carbol-fuchsin clearly showed piroplasms in different red blood corpuscles. They were approximately round in form, and lay chiefly in twos and fours in a blood corpuscle.

The four-year-old bitch above mentioned showed marked icterus on the third day of the illness. I was afterwards informed that the bitch frequently lay on the roof of the kennel in which the sick young dogs lived. Two other inmates of a kennel in the same yard remained free

from illness. The symptoms of illness disappeared in both dogs in eight days under treatment with liquor potassæ arsenitis. Further illnesses have not occurred since (October 22, 1909). Three days after the arsenical treatment I examined the urine of a patient and found it straw-coloured, clear, and limpid, and free from bile pigment, albumin, hæmoglobin, and red blood corpuscles.

(*Zeitschrift für Veterinärkunde*).

New Instruments and Appliances.

NEW MOLAR TOOTH FORCEPS.

SUGGESTED BY A. R. ROUTLEDGE, F.R.C.V.S., LOUTH.

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Books and Periodicals, &c., Received.

Proceedings of the Royal Society of Medicine; London University Gazette; The Representative (Dick College); A Handbook of Practical Parasitology, by Braun and Lühe (Bale, Sons and Danielsson, Ltd., London. 10s. 6d. net); Bulletin of The Bureau of Sleeping Sickness; The Rhodesian Agricultural Journal; The Commercial Motor.

Letters and Communications, &c.

Professol Schimmell; Dr. Stapley; Mr. A. Wilson; Mr. Bevan; Mr. G. H. Livesey; Mr. P. R. Thompson; Mr. Motton; Mr. Mayall; Board of Agriculture and Fisheries; Department of Agriculture and Technical Education for Ireland; Professor G. H. F. Nuttall.

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